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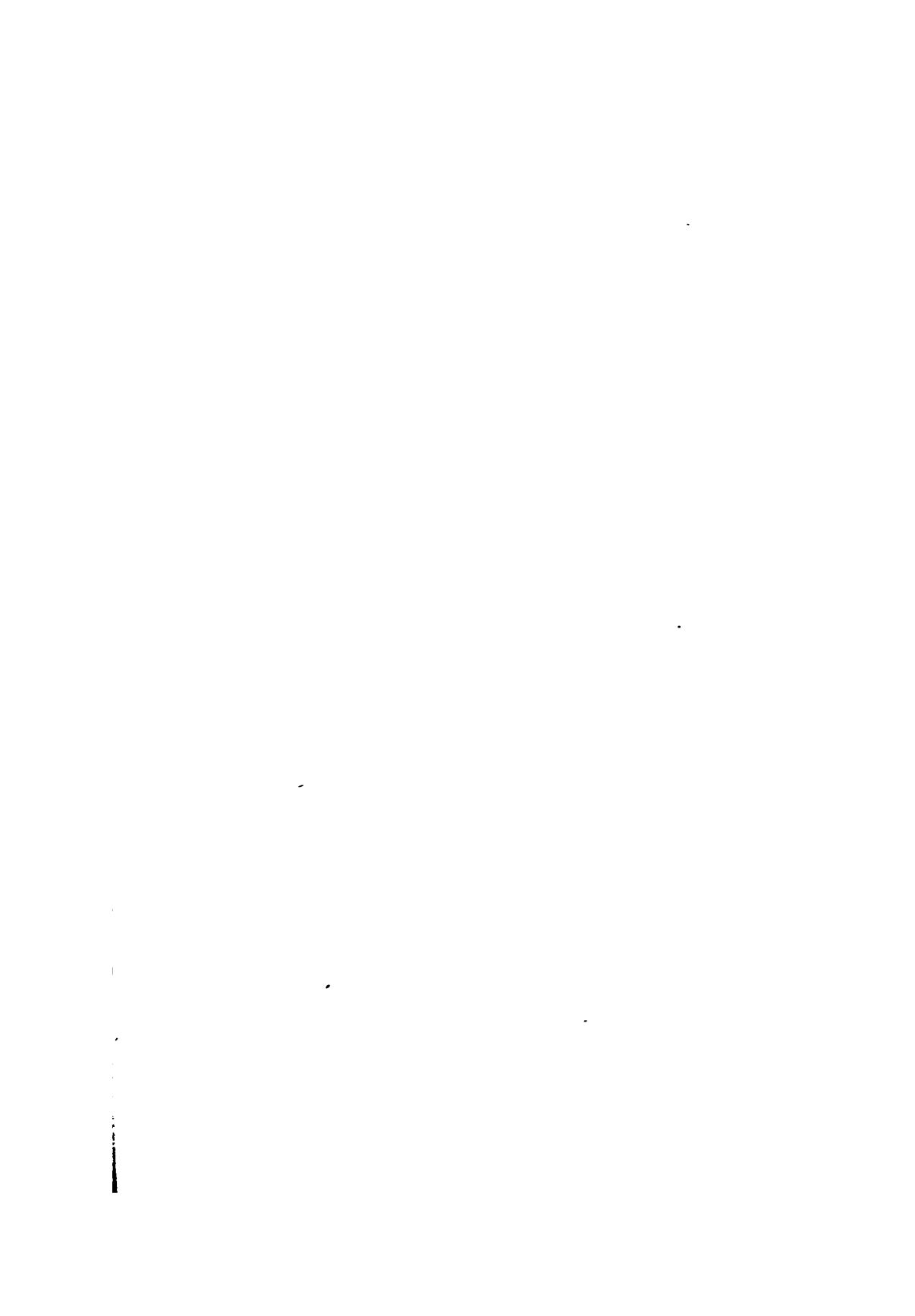


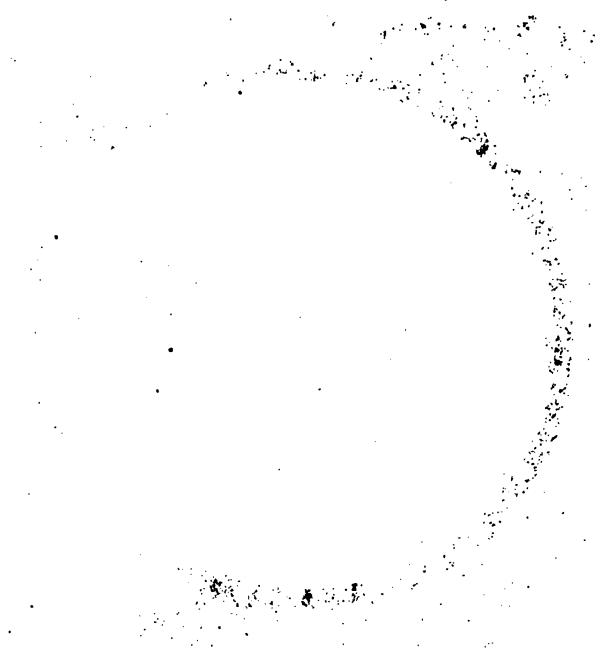


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DISEASES
OF THE
BRAIN AND SPINAL CORD.







OPTIC NEURITIS.

ANATOMY OF THE BRAIN

AND SPINAL CORD

DESIGNED TO ASSIST STUDENTS OF MEDICINE,
AND THOSE OF THE
DENTAL SURGICAL DEPARTMENT,

WITH AN INTRODUCTION TO THE STUDY OF ANATOMY,
AND THE HISTORY OF THE
ANATOMICAL SCIENCES.

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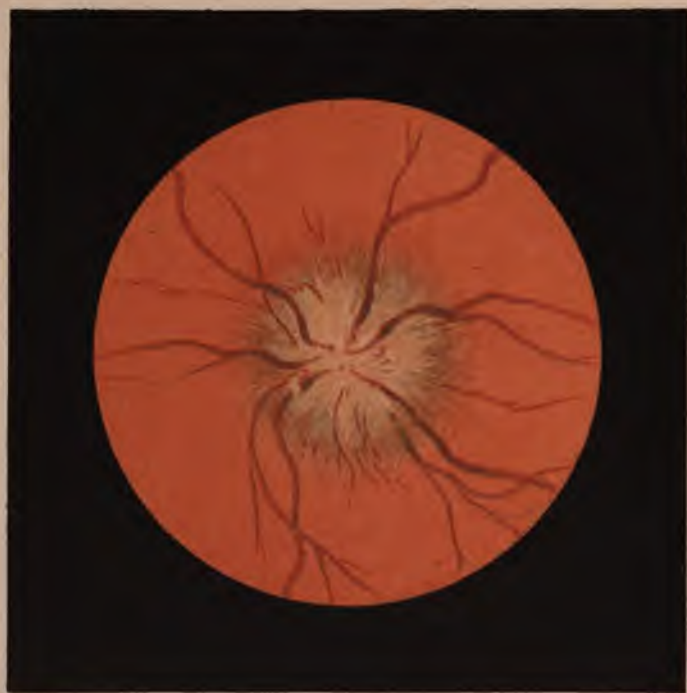
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OPTIC NEURITIS.



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AND

SPINAL CORD:

*A GUIDE TO THEIR PATHOLOGY, DIAGNOSIS,
AND TREATMENT.*

*WITH AN ANATOMICAL AND PHYSIOLOGICAL
INTRODUCTION.*

BY

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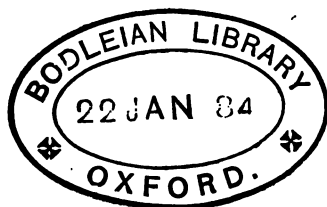
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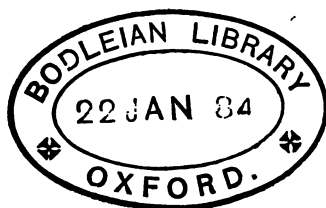
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P R E F A C E.

I VENTURE to offer this short and, I fear, very imperfect treatise to the profession, under the impression that the increasing interest taken in nervous diseases has created a demand for a work that, like the present, shall be accessible in a reasonable compass to the student, and to the busy practitioner whose engagements do not admit of the leisure necessary to master voluminous works.

It has been my aim to make the book essentially practical, and to furnish the reader with the main facts and features of the diseases of the brain and spinal cord in as concise a manner as possible. Originally written for *The Medical News*, a journal for students, it appears in its present and amended shape at the request of many professional friends and others who read it in its primitive form.

I have introduced a chapter on General Paralysis of the Insane, for which I am indebted to my friend



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Dr. T. W. McDowall, the able Medical Superintendent of the Northumberland County Asylum, Morpeth.

My thanks are due to Drs. Byrom Bramwell and Ross for their kindness in allowing me to use some of the diagrams of their respective books. Several of the illustrations in the following pages are simple pen-and-ink sketches which were produced in the present form for the sake of cheapness, so that the critic who looks for elaborate drawings will inevitably be disappointed; but at the same time it is hoped that the cuts will render the text more intelligible.

I have been largely indebted to the writings of Charcot, Erb, Bastian, Ross, Rosenthal, Bramwell, and Gowers, together with many others who have been freely laid under contribution.

DAVID DRUMMOND.

7, SAVILLE PLACE, NEWCASTLE-ON-TYNE.

October, 1883.

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DISEASES
OF THE
BRAIN AND SPINAL CORD.

THE BRAIN.

ANATOMICAL AND PHYSIOLOGICAL
INTRODUCTION.

CHAPTER I.

ANATOMY.

THE brain, or encephalon, which comprises that portion of the cerebro-spinal system contained within the cranium, is made up of various nerve centres and their connecting fibres, the former gray matter and the latter white.

This mass of nervous matter is found to weigh in the male about 50 oz., and in the female about 44 oz. But it must be understood that brains of a much lighter weight may functionate after a normal fashion. Wagner, for example, has recorded the fact that the brain of a certain celebrated mineralogist only amounted to 43 oz.

The cerebrum, composed of two large hemispheres, presents on its surface a number of well-marked convolutions (fig. 1), which are divided into groups by

the arbitrary sub-division of the hemisphere into lobes, viz., frontal, parietal, temporo-sphenoidal, occipital, and central, or island of Reil. The convolutions are lined on the surface by gray matter, an arrangement which is calculated to provide the most extensive surface while occupying the most limited space.

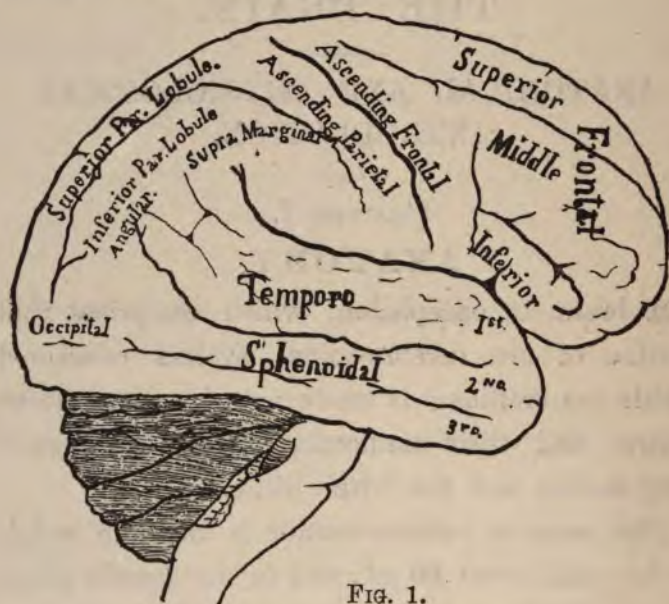


FIG. 1.

The frontal lobe, confined posteriorly by the fissure of Rolando, presents three convolutions—superior, middle, and inferior—which run from before backwards to meet the ascending frontal, a fourth and very important one which lies alongside of, and anterior to, the fissure of Rolando. It is important to recollect that the inferior frontal

convolution is intimately related at its posterior termination to the lower extremities of the ascending frontal and ascending parietal convolutions, and is closely connected with the five or six convolutions which make up the island of Reil, towards its posterior and inner aspect.

The parietal lobe, the limits of which are not so clearly defined, lies behind the fissure of Rolando, above that of Sylvius, and in front of the parieto-occipital. The convolutions which compose this lobe are:—the ascending parietal, which runs posterior to the fissure of Rolando, and is parallel to the ascending frontal, the lower end of which it joins; the superior parietal lobule, a continuation of the upper extremity of the ascending parietal convolution; and the inferior parietal lobule, situated below the intra-parietal fissure, and subdivided into the supra-marginal gyrus in front, and the angular gyrus behind.

The temporo-sphenoidal lobe, lying below the fissure of Sylvius, presents three convolutions—superior, middle, and inferior.

The occipital lobe calls for no particular description.

The central lobe, or island of Reil, is not seen on the surface of the brain, but is brought into view when the edges of the fissure of Sylvius are separated (fig. 2). It is composed of five or six convolutions which are closely associated with the

lenticular nucleus of the corresponding corpus striatum. A glance at fig. 3, which represents the upper surface of the brain, enables one to understand the general plan of the important superior and ascending convolutions. The ascending frontal communicates with the superior frontal above, and with the inferior, or third, frontal below; whilst it also joins with the ascending parietal, and that both above and below the fissure of Rolando. The

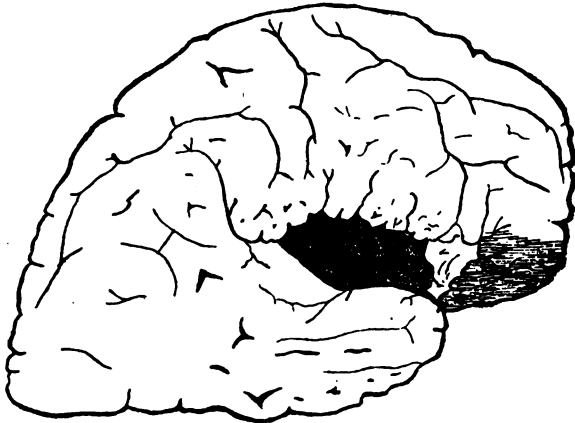


FIG. 2.

ascending parietal convolution, as it reaches the upper part of the fissure of Rolando (marked R. on the right hemisphere, fig. 3), turns backwards, and is continuous with the convolutions of the superior parietal lobule. Thus it will be seen that the convolutions which form the so-called "motor area," viz., ascending frontal, ascending parietal, posterior part of superior frontal, and the superior

parietal lobule, are intimately connected with one another. S, fig. 3, represents the upper limit of the fissure of Sylvius, whilst Inf., Mid., and Sup., are placed upon the inferior, middle, and superior frontal convolutions respectively.

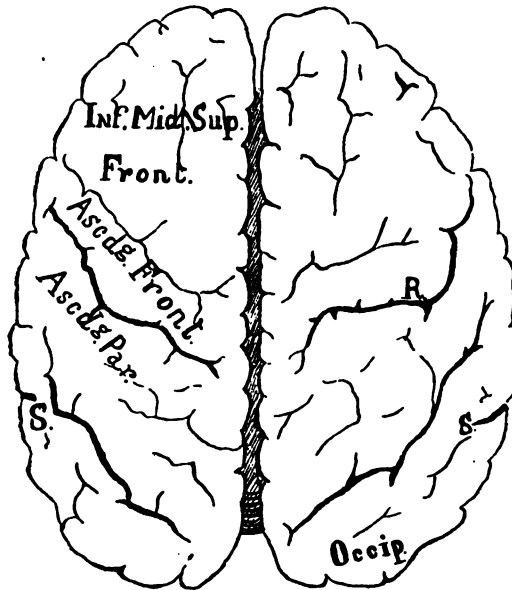


FIG., 3.

On the internal surface of the cerebrum (fig. 4) the following convolutions and sulci are plainly marked:—anteriorly and above, bordering on the edge of the hemisphere, is the marginal convolution proper; but, as it is continuous with the superior frontal convolution towards its posterior extremity, it is convenient to look upon it as the median aspect of the superior frontal (fig. 4). This convolution is

bounded below by the calloso-marginal fissure, which separates it from the gyrus fornicatus. The gyrus fornicatus, as it borders on the corpus callosum, often gets the name of the convolution of the corpus callosum. It commences near the anterior perforated spot, turns round the anterior extremity of the corpus callosum, and running along on its surface and bending down behind its posterior extremity takes the name of the uncinatè gyrus.

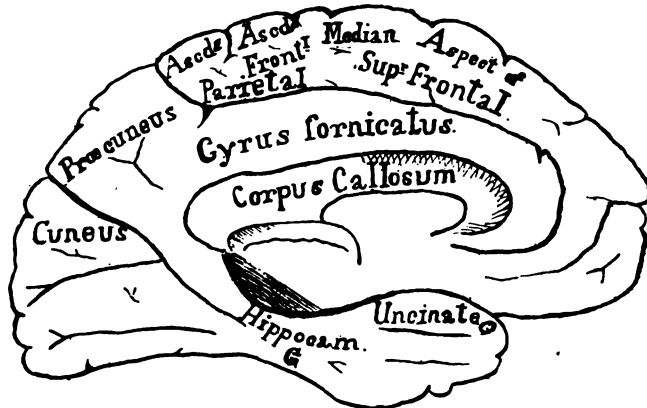


FIG. 4.

The hippocampal or uncinatè convolution or gyrus is formed by the posterior extremity of the gyrus fornicatus and a convolution from the occipital lobe (lobulus lingualis or occipito-temporalis medialis). The quadrate lobule, or præcuneus, is bounded in front by the posterior end of the calloso-marginal fissure, and behind by the parieto-occipital fissure. The cuneus is wedge-shaped, and is

bounded in front by the parieto-occipital fissure, and behind by the calcarine fissure.

The corpus callosum serves as the great transverse commissure of the brain.

RELATIONS OF THE CONVOLUTIONS TO THE SKULL.

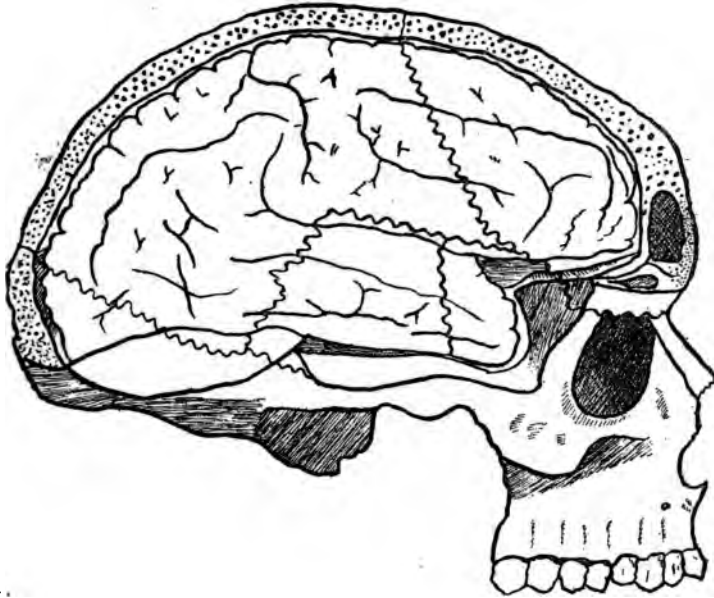


FIG. 5 (*after Turner*).

It is confessedly a difficult matter to give clear and definite lines whereby the positions of the convolutions and fissures may be marked out by an external examination of the cranium; but the following hints will be found to be of some service. I have the authority of Turner for stating that, by manipulating the head, one can generally recognise

the coronal and lambdoidal sutures, the parietal eminence, and the external angular process of the frontal bone. A line joining the latter point (external angular process of the frontal) and the parietal eminence, and passing through the lower extremity of the coronal suture, will be found to correspond tolerably accurately with the fissure of Sylvius; the fissure commencing anteriorly at the point where this line strikes the coronal suture, and ending posteriorly just in front of the parietal eminence. The fissure of Rolando runs nearly parallel to the coronal suture, and will be found to lie about half-way between that suture and the parietal eminence; or, according to Turner, the fissure of Rolando is situated at a distance behind the coronal suture varying from two inches to an inch and a half. The parietal eminence marks the position of the supra-marginal gyrus (fig. 1). The occipital lobe lies between the lambdoidal suture and the occipital protuberance—a point which can be recognised with ease. Acting upon these brief particulars, the situations of the principal convolutions may be mapped out with a moderate degree of accuracy.

CRANIAL NERVES.

At the outset we must familiarise ourselves with the relation of these nerves to the different parts of the brain at the base, and to this end must be acquainted with their superficial origins, so as to

possess landmarks which shall materially aid us in the localisation of brain lesions.

Fig. 6 is a drawing of the base of the brain, and shows the superficial origins of the cranial nerves.

The first, or olfactory nerve, starts from the anterior perforated space, and passes forward in a groove on the under surface of the frontal lobe.

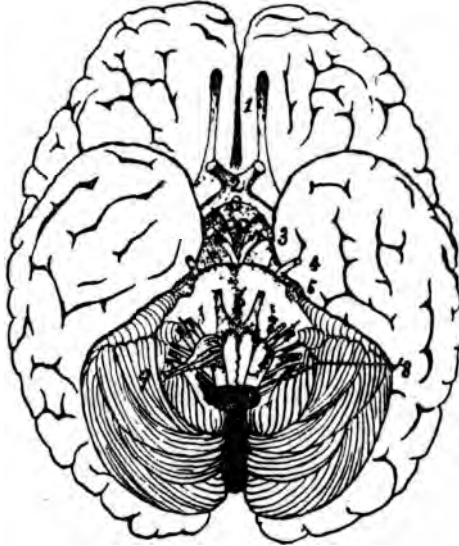


FIG. 6.

The second, or optic nerve, starts from the optic commissure, and, traced backwards, the optic tract embraces the crus cerebri on each side.

The third, or motor oculi, has its apparent origin from the inner surface of the crus, immediately in front of the pons.

10 *Cranial Nerves—Superficial Origins.*

The fourth, or patheticus, appears at the outer side of the crus at its posterior extremity, in front of the pons; though it really leaves the brain substance at the upper part of the valve of Vieussens behind the corpora quadrigemina.

The fifth, or trigeminus, arises by two roots from the side of the pons.

The sixth, or abducens, passes out between the upper end of the anterior pyramid of the medulla and the posterior border of the pons.

The seventh—made up of the facial, or portio dura, and the auditory, or portio mollis—arises from the medulla oblongata in the groove between the olivary and restiform bodies close to the pons. The facial is anterior to the auditory.

The eighth consists of three nerves, viz., the glosso-pharyngeal, pneumogastric, and spinal accessory. These arise from the side of the medulla behind the olivary body, the glosso-pharyngeal above, the spinal accessory below, and the pneumogastric between these two. Some of the fibres of the spinal accessory arise from the upper part of the cord in common with the motor roots of some of the cervical nerves.

The ninth, or hypoglossal, arises by ten or twelve filaments from the groove between the anterior pyramid and the olivary body.

Such, then, are the points of importance to be observed on the external surface of the brain. The

basal ganglia and ventricles of the brain are exposed to view in fig. 7 (*after Hirschfeld*) by the removal of the upper part of both hemispheres. At first, before the level of the corpus callosum is reached, the exposed white matter on each side, which is surrounded by a layer of gray matter, is called the

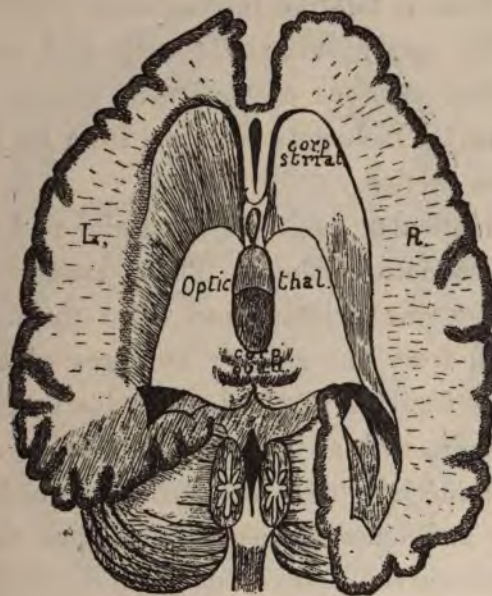


FIG. 7.

centrum ovale minus; but when the cut hemisphere surfaces are on the level of the corpus callosum the wide expanse of white matter gets the name of the centrum ovale majus. On removal of the corpus callosum—the connecting medium of the hemispheres—the lateral ventricles are exposed. An-

teriorly the septum lucidum divides these ventricles; posteriorly they are separated by the third ventricle, behind which are situated the corpora quadrigemina. Within the lateral ventricles are found the corpora striata in front, and the optic thalami behind. Each corpus striatum presents an intra-ventricular portion, or the caudate nucleus (so named from a tail-like prolongation which is seen when the upper surface is cut), and an extra-ventricular portion, or lenticular nucleus (fig. 8, *Len. nuc.*), which gets its name from its lens-like shape on section.

The lenticular nucleus is posterior and external to the caudate nucleus. These masses of gray matter—corpus striatum and optic thalamus—are separated from one another by fibres which make their way from the cerebral peduncles to the cortex of the brain, and, as they spread upwards and outwards bordering upon the lenticular nucleus and anterior and outer surface of the optic thalamus, are called the *internal capsule*. In fig. 7 the upper and inner portion of the corpus striatum (left side) has been removed to expose the spreading peduncular fibres. The fourth ventricle is shown in the same figure, a portion of the cerebellum having been removed for that purpose.

Fig. 8 (*after Hirschfeld*) represents the base of the brain, parts of which have been removed to expose on the right side (R.) the lenticular nucleus of

the corpus striatum. The right crus, or peduncle, can be seen to pass up beneath the optic tract to the lenticular nucleus, where the fibres spread, and in radiating separate it from the caudate nucleus, as is shown on the left side (L.); for here the lenticular nucleus is removed to expose these spreading fibres,



FIG. 8.

which are continuous with the anterior pyramids of the medulla through the pons, fig. 8 (portion of pons removed). Thus the internal capsule separates the caudate from the lenticular nucleus, and the latter from the optic thalamus. The narrow band of gray substance which lies adjacent to the lenti-

cular nucleus, fig. 8 (left side), is called the claustrum; the white fibres which separate the claustrum from the lenticular nucleus get the name of the external capsule.

It will thus be apparent that the nerve fibres which connect the sensory surfaces and muscles with the gray matter of the cerebral convolutions (highest

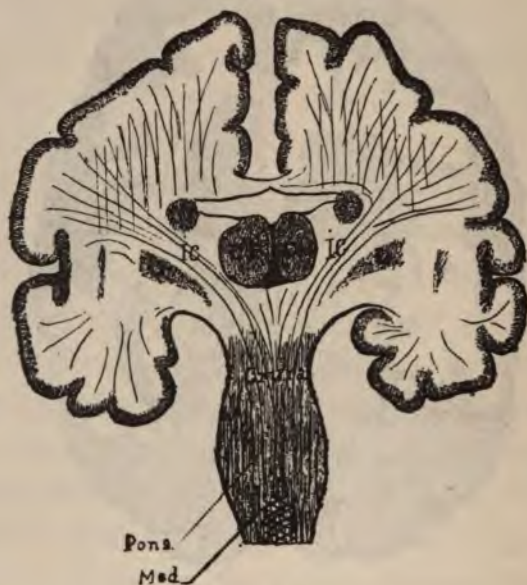


FIG. 9.

centres) are very closely related to the large ganglia at the base of the brain—the corpora striata and optic thalami. Fig. 9, which is a diagram of the brain in transverse vertical section, shows the connection existing between the peduncular fibres and these ganglia. Having passed up through

Internal Capsule—Pons and Medulla Oblongata. 15

the pons the fibres enter the crura, and then form themselves into the internal capsule (i. c., fig. 9), which is a fan-shaped layer of white fibres; by this layer the caudate nucleus of the corpus striatum and the optic thalamus (Op. Th.), are separated from the lenticular nucleus (L.), the former lying above and inside, and the latter below and external to the internal capsule. The fibres then diverging into the corona radiata pass on to the great mass of gray matter which is spread over the cortical convolutions. The fibres of the internal capsule, or rather their anterior division, may be said to pass directly through the corpus striatum, whilst the posterior division borders on the optic thalamus. It should be recollected that the term corpus striatum is often confined to the intraventricular portion or caudate nucleus.

The pons varolii and medulla oblongata are shown in fig. 10. The superficial transverse fibres of the pons are cut away on the left side (L.) to expose the anterior or motor (M.), which pass up from the anterior pyramids (A. P.) of the medulla, and enter the crura of the crus to form that portion of the internal capsule which separates the caudate from the lenticular nucleus. On the right side (R.) the deeper transverse fibres are removed to show the deeper longitudinal ones, probably sensory (S.); these latter form the tegmentum of the crus and border on the optic thalamus in their passage

upwards. The right anterior pyramid is cut to display the right olivary body (O. B.), the left olivary body being hidden partially by the left anterior pyramid (A. P.), which is left intact in the figure. The decussation of the anterior pyramids is also shown in fig. 10 (Dec.). But

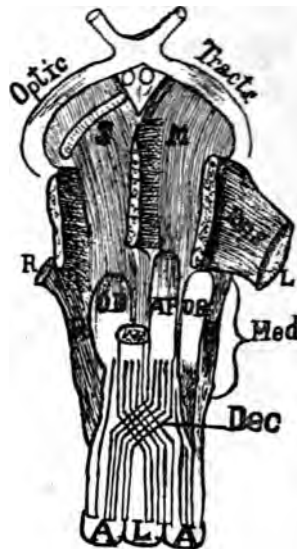


FIG. 10.

it must be observed that portions of the anterior pyramids do not decussate, but pass to the anterior columns of the same side (A. A.). The decussating fibres pass to the lateral columns of the cord marked (L.).* Thus the medulla is composed of

* The letter (L.) is placed in the middle for convenience.

posterior and anterior pyramids, which are bounded on each side by the olivary bodies, as these in turn are similarly bounded by the restiform (R. R.). Fig. 10 is also from a plate in Hirschfeld's Atlas, but altered. In figs. 11 and 12 the deep origins of the cranial nerves are represented. These figures

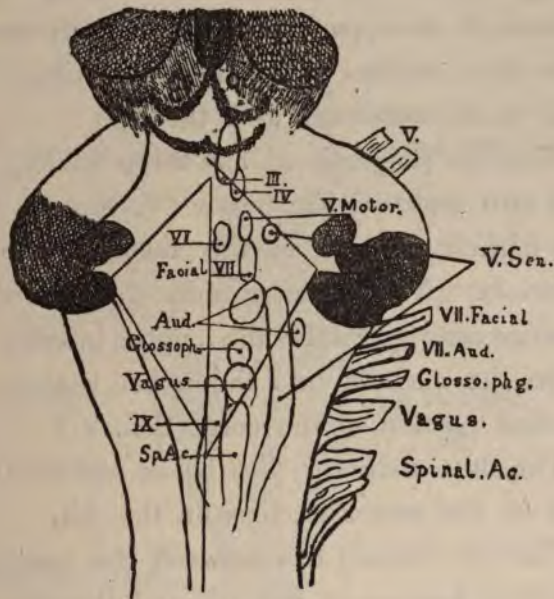


FIG. 11.

are altered from diagrams illustrating Erb's article on the medulla in Ziemssen's Cyclopædia, vol. xiii. Fig. 11 is a transparent view of the medulla from behind; on the right side the nerve-nuclei are diagrammatically represented. At the top of the diagram are placed the corpora quadrigemina (C. Q.), which lie on the top of the posterior

part of the cerebral peduncles and immediately behind the third ventricle. The gray matter from which the 3rd cranial nerve (motor oculi) arises is situated beneath the corpora quadrigemina, and extends down to the upper extremity of the fourth ventricle (the lozenge-shaped space shown in fig. 11), and at its lower extremity is closely related to the upper nucleus of the 5th nerve (V.). The chief nucleus of the 4th (patheticus) may be said to be connected with the 3rd.

The 5th (trigeminus) has three nuclei, a motor and two sensory; the motor (V. motor) occupies the highest position in the floor of the fourth ventricle. To its outer side lies the short or superior sensory, whilst the long or inferior extends down the medulla into the upper regions of the cervical portion of the cord (Sen. V.).

The 6th (abducens) lies below and to the inner side of the motor nucleus of the 5th.

The 7th (facial) lies between the upper part of the long nucleus of the 5th and the 6th.

The 7th (auditory) is supposed to have four nuclei, but for convenience I have only inserted the two central ones (the so-called anterior and posterior median acoustic nuclei).

The 8th has three nuclei: the superior from which the glosso-pharyngeal arises (Glosso.); the middle for the vagus; and the inferior (Sp. Ac.) giving origin to the spinal accessory, and extending

down, like the inferior sensory of the 5th, into the cervical cord.

The 9th (hypoglossal) lies to the inner side of these (8th) nuclei, and is also a long nucleus, though it is practically confined to the limits of the medulla. Fig. 12, which is a transparent lateral view of the medulla, shows the relative positions

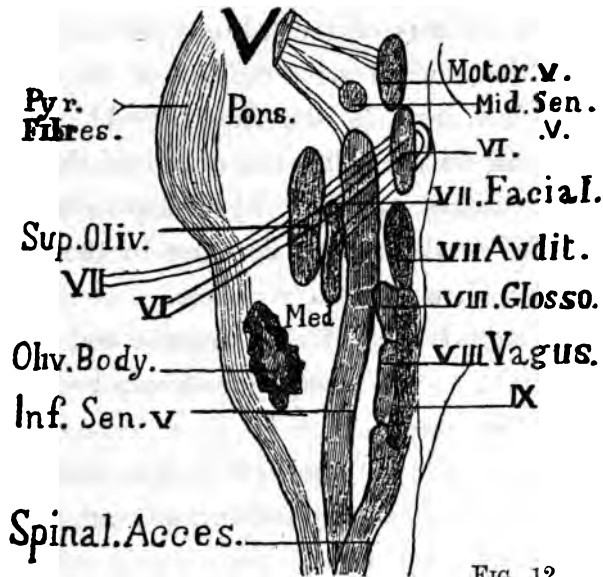


FIG. 12.

of the nuclei, and represents the right half of the medulla seen from the surface of section. The nuclei which lie closer to the cut surface—i.e., to the median line—are deeper shaded than those more remote.

To the left of the figure are the pyramidal fibres (Pyr. fibres), which are continuous with the anterior

pyramids of the medulla, and therefore are situated in the anterior part of the pons and medulla. The nerve fibres of the 5th, 6th, and 7th (facial) are given (V., VI., and VII.). The fibres of the facial first pass backwards to the outside of the nucleus of the 6th, and then passing with a bend forwards appear in the groove between the olivary and restiform bodies immediately behind the pons. The motor nucleus of the 5th occupies a position close to the surface of the floor of the fourth ventricle, but more to the side (fig. 11). From fig. 12 it will be seen that the nuclei of the sixth, posterior median acoustic, hypoglossal, and the three nuclei of the eighth, lie close to the surface in the floor of the fourth ventricle.

The olivary bodies of the medulla and pons—the latter the so-called superior olivary body—are shown in the figure.

The deep origins of the first and second nerves lie out of the range of the diagrams, but may be briefly referred to here.

The first (olfactory) has been traced “to a nucleus in the substance of the temporo-sphenoidal lobe, in front of the anterior extremity of the hippocampus” (Rolando, Luys, Foville).*

The origin of the second (optic) has been variously stated, but perhaps the most important

* Quain's “Anatomy,” viii. ed., p. 566.

fibres arise from the nates or the anterior of the corpora quadrigemina, though it must be recollected that many of the fibres have been traced to cells which occupy the lower stratum of the optic thalamus.

THE CEREBELLUM.

The cerebellum, like the cerebrum, is composed of white and gray matter; the latter, with the exception of the corpus dentatum, is arranged on the surface of the minute laminæ or convolutions of which the cortical portion of the organ is formed. The white matter serves to connect the gray with the cerebro-spinal axis. This connection is made by means of the cerebellar peduncles. The superior, or *processus e cerebello ad corpora quadrigemina*, pass up to the corpora quadrigemina, and so on by the crura into the cerebrum. The middle connect the hemispheres by fibres passing round the under surface of the pons in a transverse direction, so that the cerebellum may be said to embrace the pons by its middle peduncles. The inferior, formed by the restiform bodies, unite the cerebellum with the medulla and cord.

CIRCULATION OF THE BRAIN.

The importance of being familiar with the cerebral circulation will be apparent from a consideration of its obvious relation to cerebral pathology. In illustration take the dependence

of the great majority of cases of paralysis from brain disease upon vascular changes. Blood is supplied to the brain by two sets of vessels, the vertebral and the internal carotid arteries. Fig. 13

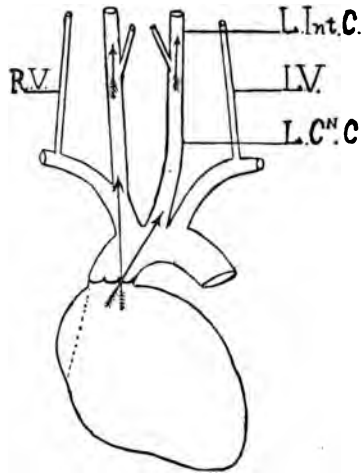


FIG. 13.

is a *schema* of these vessels, and shows their relation to the larger vessels and the heart. The vertebrals (on the left side marked L. V.) come off from the subclavian arteries at right angles, whilst the internal carotids (on the left side marked L. Int. C.) are almost a direct continuation with the first part of the aorta, and therefore may be said to be in the direct main-stream. This point is important, for not only are these vessels more subjected to rapid variations in blood tension, but from their position and size enable embola (fibrinous masses), when set free from the aortic valves or other sites

where such deposit is wont to occur, to reach the brain in the course indicated by the arrows in the figure rather than by the vertebrals. The internal carotid and vertebral arteries, having entered the cranium, unite to form at the base of the brain the circle of Willis (fig. 14) in the following manner:—the vertebrals (V.) join at the upper

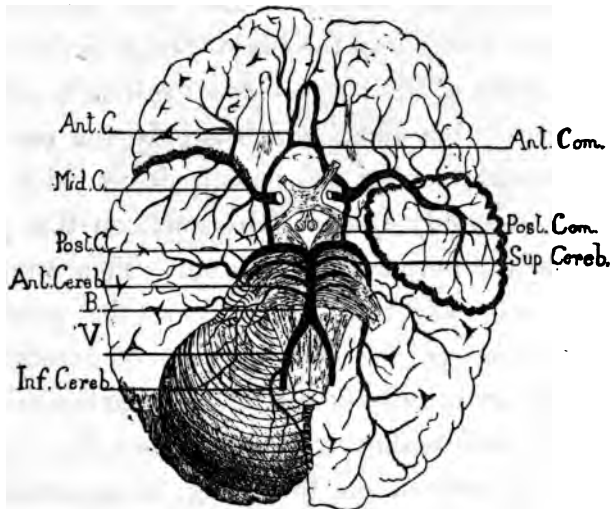


FIG. 14.

part of the medulla to constitute the basilar artery (B.). This vessel divides into the posterior cerebral arteries (Post. C.) from which the posterior communicating (Post. Com.) pass to join the internal carotids. The anterior cerebral arteries from the internal carotids (Ant. C.) are united by the anterior communicating (Ant. Com.). These with the middle cerebral (Mid. C.) practically supply the cerebrum with blood.

The cerebellum is supplied by three sets of vessels:—the inferior cerebellar (Inf. Cereb.), which arises from the vertebral; the anterior cerebellar (Ant. Cereb.), from the basilar; and the superior cerebellar (Sup. Cereb.), from the same vessel just before it divides into the posterior cerebral arteries.

It is almost an established fact that the arteries which pass direct into the brain-substance from the large arteries at the base—“*basal system*”—do not anastomose; but those which supply the convolutions would appear to communicate with one another tolerably freely. However, on this point there is a difference of opinion. This want of inosculation—affecting, as it does, the most important parts at the base—should be recollected. The chief arteries to be attended to are the middle, anterior, and posterior cerebrals, and the basilar. On the left side of the brain (fig. 14) a portion of the middle lobe is removed to show the distribution of the middle cerebral artery, which, coming off from the internal carotid, of which, indeed, it is almost a direct continuation, passes outwards to enter the fissure of Sylvius. Some branches pass from it to the anterior or frontal lobe, and others to the middle or temporo-sphenoidal. The anterior cerebral passes forwards from the internal carotid, and bends round the corpus callosum, sending branches to the under surface of the frontal

lobe. In fig. 15 this vessel is represented as coursing on the median aspect of the brain, where it supplies through its branches the marginal convolution, gyrus fornicatus, and corpus callosum, with



FIG. 15.

the præcuneus and cuneus. In fig. 15 the vertebral (V.), basilar (B.), internal carotid (Int. C.), and middle cerebral (Mid. C.), are also shown.

Fig. 16, which is a transparent view of the base of the brain, shows diagrammatically the distribution of the principal vessels. The basal ganglia—corpus striatum and optic thalamus, corpora quadrigemina, and island of Reil, are represented in the figure by dotted lines thus:—the caudate nucleus of the corpus striatum (Corp. S.), the lenticular nucleus (L.), the optic thalamus (Op. T.), the corpora quadrigemina (C. Q.), the island of Reil (Is. R.). The anterior cerebral supplies the frontal lobe and

most of the caudate nucleus. The middle cerebral supplies the lenticular nucleus, part of the caudate nucleus, and the anterior and external portions

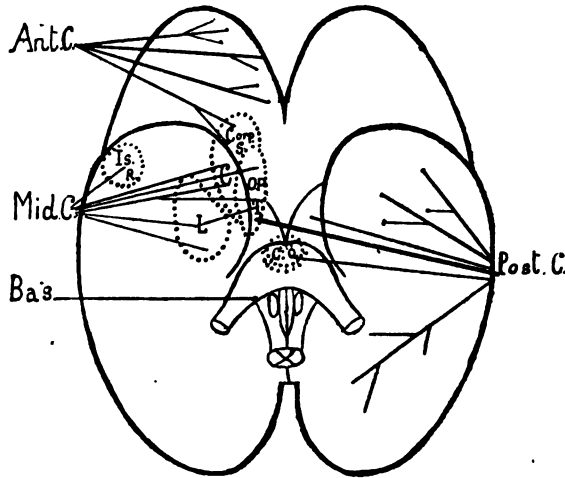


FIG. 16.

of the optic thalamus. The posterior cerebral nourishes the posterior part of the optic thalamus, the crus, corpora quadrigemina, and the postero-inferior part of the cerebrum. The pons is supplied by the basilar.

In fig. 17 the blood supply to the principal convolutions on the outer surface of the brain is shown; the anterior cerebral furnishing the first and second and the upper part of the ascending frontal with vessels. The middle cerebral supplies the third or inferior frontal, the lower part of the ascending

frontal, the ascending parietal, the superior parietal lobule, the supra-marginal, the angular gyri, and the first temporo-sphenoidal convolution. The posterior cerebral sends branches to the occipital and the inferior part of the temporo-sphenoidal lobes.

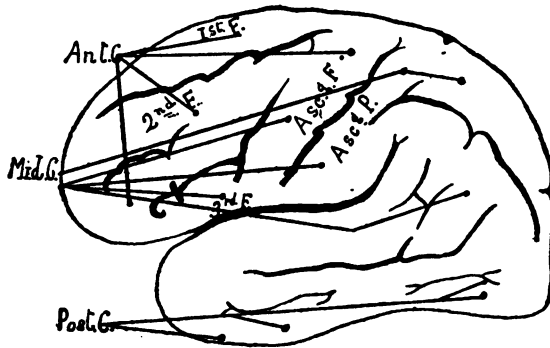


FIG. 17.

In describing the circulation I purposely omit the venous channels for two reasons—first, because the details of the subject are clearly set out in every anatomical text-book; and, secondly, because their importance in relation to clinical work is of less degree, and scarcely commensurate with the space and time necessary for their consideration.

MEMBRANES.

The *pia mater* is a delicate but exceedingly vascular membrane, and gives off to the cortical gray matter of the brain, to which it is very intimately applied, a rich supply of minute vessels. It invests

the entire surface of the brain, dipping down into the sulci between the convolutions.

The arachnoid is the so-called serous covering of the brain which invests it outside the pia mater. It passes over the eminences of the convolutions without dipping down into the sulci. Between the arachnoid and the pia mater is the sub-arachnoid space which contains the sub-arachnoid fluid. The view that the arachnoid is a shut sac with its visceral and parietal layers, the latter being reflected on to the dura mater, is now generally discarded.

The dura mater is a dense fibrous membrane of great strength lining the inner surface of the skull, and therefore forming the internal periosteum of the bones which compose it. Its internal surface is exceedingly smooth, being lined by pavement epithelium. The dura mater, with the processes which pass from it into the interior of the cranium between the hemispheres, and also at other situations, serve to support and protect the brain.

CHAPTER II.

INTRODUCTION (continued).

THE FUNCTIONS OF THE BRAIN.

THE account I have given of the structure of the brain, although brief, should have cleared the way for, and rendered easy, the consideration of its

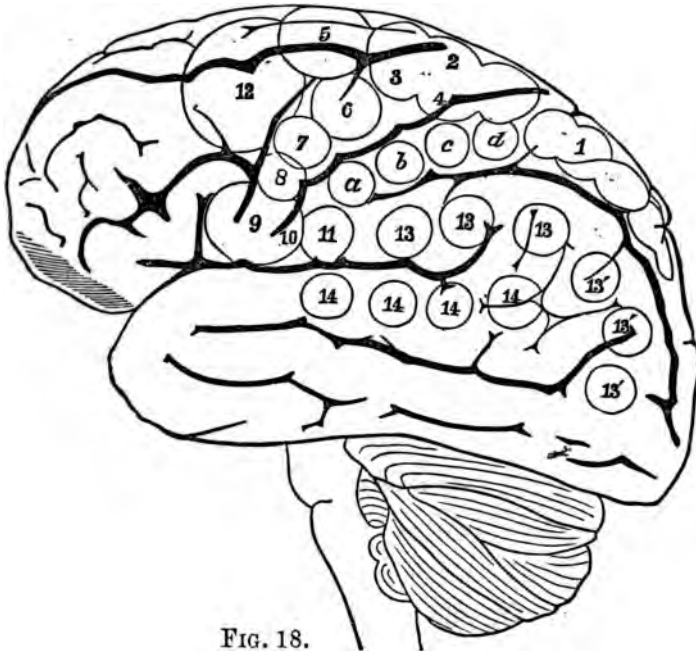


FIG. 18.

functions, and of such opinions thereon as are well grounded yet not entirely or universally accepted.

It will be convenient to commence with the cortical centres, a knowledge of which, as ex-

pounded by Ferrier and others, has become—at all events as regards some of them—essential for the proper elucidation of many of the mysteries of brain disease. In fig. 18, which is a side view of the left half of the brain (after Ferrier), certain areas are marked by Ferrier in conformity with the results he obtained by experimenting on the brains of monkeys.

(1) On the superior parietal lobule, the centre for the opposite leg.

(2, 3, 4) On the ascending frontal, ascending parietal, and superior frontal convolutions, for certain movements of opposite arm and leg—*e.g.*, as in swimming.

(5) On the posterior parts of superior and middle frontal convolutions, the centre for the opposite arm and hand—extension forwards.

(6) Where the ascending frontal unites with the middle frontal, for supination and flexion of the opposite forearm.

(7 and 8) On lower part of the ascending frontal, for certain movements of the muscles of the mouth (opposite side).

(9 and 10) On the posterior part of the inferior frontal and the lowest part of the ascending frontal, Broca's convolution, centres for mouth and tongue—so called area of ataxic aphasia.

(11) Also for movements of the mouth—*i.e.*, retraction of the angle.

(12) On the posterior parts of the superior and middle frontal convolutions, the centre for lateral movements of the head and eyes.

(13, 13, 13, 13', 13', 13') On the supra-marginal and angular gyri, centre for vision and certain movements of the eyes, (13) upwards, and to the opposite side, (13') downwards, and to the opposite side.

(14, 14, 14, 14) On the superior temporo-sphenoidal convolution, centre for hearing, and for movements of the eyes, head, and ears.

(a, b, c, d) Ascending parietal convolution, for movements of the opposite hand and wrist.

Limited space and the elementary character of this work admit only of a statement of Ferrier's conclusions, and of my adoption of them in the main as the result of considerable clinical experience in cerebral disease.

Fig. 19 represents in diagrammatic form the connection between some of the chief cortical centres and the extremities; the hemispheres of the brain are, as it were, separated in front in order to show more clearly the position of the fibres in relation to the corpus striatum. It will be seen that the motor fibres on leaving the motor centres in the cortex pass down to the corpus striatum, where they form themselves into the internal capsule; here they pass between the intra-ventricular and extra-ventricular portions of the corpus striatum; then running on

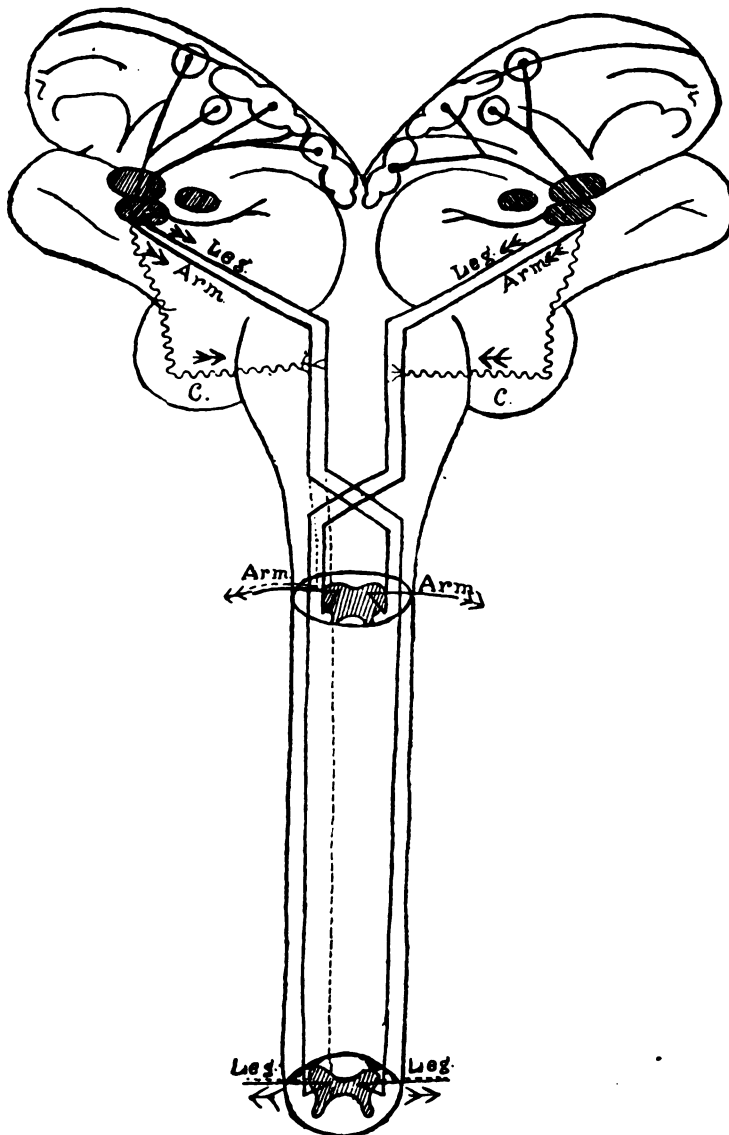


FIG. 19.

Diagrammatic representation of the Connection between the Cortical Centres and the Extremities.

into the crus, they enter the pons and medulla, in the front of which most of them decussate to the opposite side and descend the cord to supply the muscles of the side of the body opposite to the hemisphere from which they started. Others, however (left side of fig. 19, dotted line), do not decussate, but are found to continue their course through the anterior columns of the cord to the muscles of the same side of the body. On the other hand, the sensory fibres, having decussated in the cord at a place adjacent to the point of entrance (a fact which will be referred to when the structure of the cord is being considered), pass up to the opposite side of the cerebrum along the posterior part of the crus, forming the posterior part of the internal capsule and bordering on the optic thalamus, from which body they are supposed by Ferrier to radiate to the hippocampal and uncinate gyri.

In fig. 19 the wavy or sinuous line is intended to show the connection between the motor tracts from the corpus striatum and the cerebellum (C.), and the relation between that body and the cord.

Hitherto I have described the motor and sensory tracts as though they *passed by* the basal ganglia (corpora striata and optic thalami) in ascending to the convolutions. But although it is correct to say that many of the fibres do not enter into and communicate with these bodies, but pass directly to the surface, it is also, in all proba-

bility, a fact that many pass into the basal ganglia and become connected with their cells, from which fibres pass out to the gray matter; so that in many cases the cord tracts are only indirectly connected with the convolutions. Of such fibres the motor pass into the corpora striata, and the sensory into the optic thalami.

As regards the functions of the corpora striata and optic thalami, little or nothing can be advanced beyond what is furnished to us in a suggestive manner by our knowledge of the relation of the motor and sensory fibres to these bodies. This anatomical evidence, aided by pathological research, gives rise to the belief that the corpus striatum mediates in some way between the cortical motor centres and muscular movements on the opposite side of the body, and that the optic thalamus stands in a somewhat similar relation to the peripheral sensory surfaces and the cortical perceptive centres. It must be understood, however, that these vague statements have not yet been clearly proved.

It is a well-ascertained fact that in the case of some of the lower animals the most complicated movements may be performed, so long as the requisite stimuli are applied, even in absence of the cerebrum, corpora striata, and optic thalami. Thus the frog, after these parts have been removed, will swim when placed in water, croak when its flanks are stroked, and crawl up a board to maintain its equilibrium as

the board is being gently tilted ; but these actions no longer take place when the brainless frog has also been deprived of its pons, optic lobes (corpora quadrigemina), cerebellum, and medulla. These facts prove that one or more of the bodies just mentioned are capable, on being stimulated in a certain way, of transmitting co-ordinate motor impulses of the most complicated character which can excite movements in an orderly fashion, but that they cannot originate motor impulses apart from the afferent (sensory) nerves, on the one hand, and the higher cerebral gray matter (will), on the other.

PONS, AND CRURA CEREBRI.

Undoubtedly these organs are but ill understood. That they play an important part in relation to the co-ordination of movements cannot be denied ; but in the light of our present knowledge few would care to go beyond an assertion of the important part played by them in relation to the co-ordination of movements, and a recognition of their patent properties as conductors of sensory and motor impulses to and from the cerebrum. The diagnosis of lesions affecting the pons and crura is to be made upon data which are found to depend upon the relation of certain cranial nerves to these bodies, and not so much from any interference with their functions as nerve centres.

Our information regarding the *forced movements*

36 *Functions of the Corpora Quadrigemina.*

which result from injury to the pons and crura is still very vague. Section of one crus, or unilateral section of the pons, gives rise to rotation of the animal round the longitudinal axis of its body, or the animal so injured may execute other *forced movements*—*e.g.*, it may move round in a circle. But this latter—"circus movement"—results more frequently from injuries to the corpora striata and optic thalami.

CORPORA QUADRIGEMINA.

The "brainless frog" and other experiments, along with pathological research, teach us that these bodies preside in a great measure over the general movements of the body as co-ordinating centres. Very important functions in relation to the movements of the eye-balls and pupils are exercised by the nates, or anterior divisions, as co-ordinating centres. These are intimately associated together, so that when the eyes are accommodated for a near object (*i.e.*, downwards and inwards) the pupils are at the same time contracted, and on the return to parallelism the pupils dilate. In addition to their functions as centres for the co-ordination of movements, the corpora quadrigemina are very importantly related to sight. Flourens has shown that unilateral destruction of these bodies in mammals produces blindness of the opposite eye. It may be said, perhaps, that the corpora quadrigemina mediate

in relation to *sight* between the eyes and the cortical visual centres, just as the corpora striata mediate in relation to *movements* between the convolutions and the muscles. *Forced movements* have been observed as a result of injury of one side of the corpora quadrigemina.

CEREBELLUM.

The cerebellum is perhaps the most important organ of co-ordination in the brain. This conclusion is well-nigh indisputable so far as experimentation upon mammalia and birds can prove it; but it must be freely confessed that up to the present the pathological observations upon man are somewhat contradictory. In other words, whilst it is true that many cases of cerebellar disease can be diagnosed with positive certainty by the tottering, reeling gait, it is also true that the cerebellum is occasionally found extensively diseased without any such evidence of inco-ordination having been observed during life. The cerebellum seems to be the prime mover in the maintenance of equilibrium, playing a most important part in relation to station and locomotion.

MEDULLA OBLONGATA.

The great importance of, and numerous functions performed by, this organ will be apparent when it is considered that it contains a large number of nerve nuclei. Many of the functions of the medulla

38 *Centres in the Medulla—Cranial Nerves.*

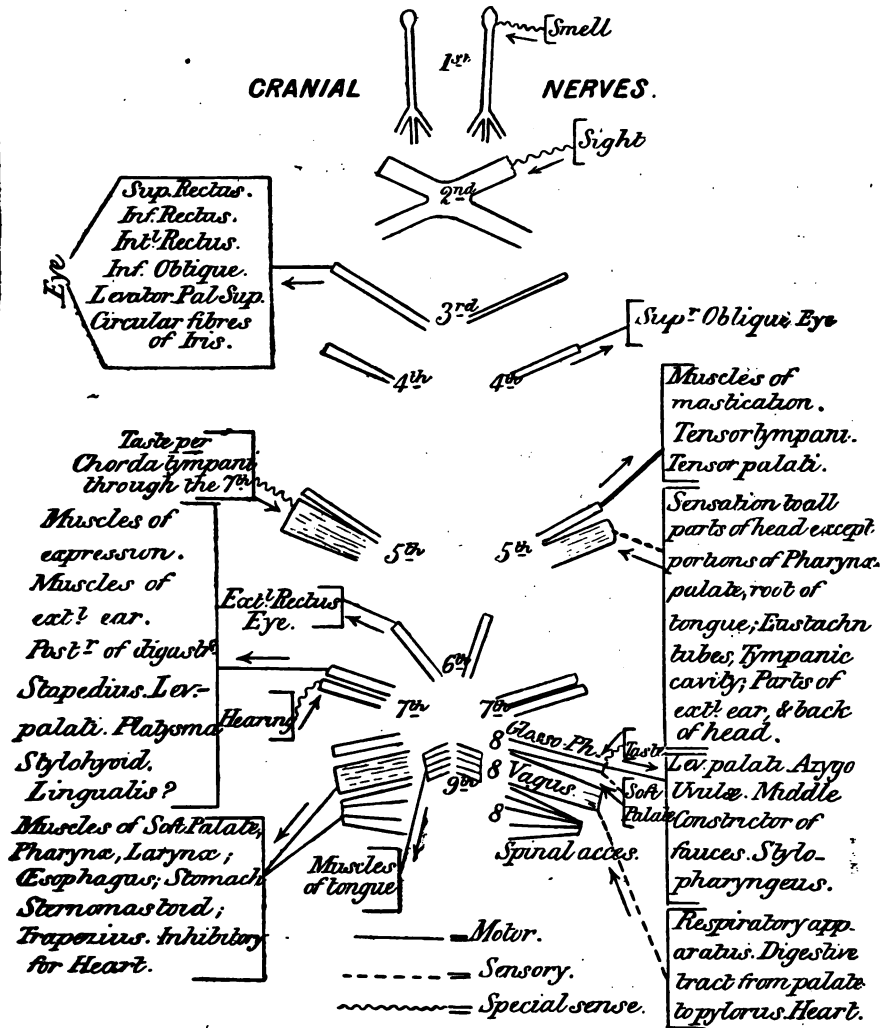
are of a vital character. Briefly, the following centres are situated in it:—1, vaso-motor, with the adjacent diabetic centre; 2, respiratory; 3, cardio-inhibitory; 4, centre for deglutition; 5, centre for articulate speech (?); and others of less importance. In connection with articulation, it should be recollected that a close anatomical and pathological relation exists between the nuclei of the nerves which supply the tongue, palate, lips, and laryngeal muscles—hypoglossal, glosso-pharyngeal, facial, and spinal accessory; the evidence of disease of these nuclei being distinct and easily recognised, marking a lesion of the medulla—bulbar paralysis.

CRANIAL NERVES.

The cranial nerves furnish the clinical physician with much that is essential from a diagnostic point of view. A glance at the accompanying table, which is arranged in a diagrammatic form, will remind the reader of the functions of these nerves.

The olfactory endow the mucous membrane of the nose only with the special sense of smell, but the trigeminus (5th), as the nerve of common sensation, is responsible for reflex phenomena, sensitiveness to the irritation of pungent vapours, pain, tactile sensation, &c. In a case of simple anosmia (loss of the sense of smell) the common sensation of the nasal fossæ is preserved.

The second or optic has received a consider-



Diagrammatic representation of the Cranial Nerves and their Functions.

able amount of attention from physiologists, with a view to determine whether or not the fibres decussate completely in the chiasma. The least assailable view appears to be that in man the decussation is not complete, though almost so. Some fibres have been observed by Henle and others to pass along the outer side of the chiasma from the quadrigeminal body to the eye of the same side; whilst a few fibres connect the eye-balls, passing from one optic nerve to the other along the front of the chiasma.

The third nerve is exclusively motor. When it is paralysed the following phenomena are observed:—external strabismus, a general immobility of the eye-ball, dilated pupil, and ptosis (drooping of the upper eyelid).

The fourth is distributed exclusively to the superior oblique muscle of the eye-ball. Very slight deviation is observed in cases of paralysis of this nerve, double vision being the most marked symptom.

The fifth has an extensive distribution. It is the great sensory nerve of the head. It is supposed to furnish the sense of taste to the anterior portion of the tongue; but many are of opinion that the facial provides the fifth with its taste fibres through the chorda tympani. The fifth has also a special motor root.

The sixth, like the fourth, is distributed to a

single ocular muscle—the external rectus. Division or disease of this nerve induces an external strabismus.

The seventh, facial, is the motor nerve for the muscles of expression. In paralysis of the right facial nerve, during energetic facial movement, the right side of the face is smooth and the eye open and staring. Deviation of the uvula, deafness, and loss of taste may also be observed according to the position of the lesion, and depend upon the fact that the seventh, facial, sends filaments in its course through the aqueduct of Fallopius to the levator palati (great superficial petrosal), the stapedius, and, as referred to above, supplies taste to the tip of the tongue through the chorda tympani. I may here parenthetically remind the reader that salines and sweets are appreciated by the tip of the tongue only, and bitters by the back of the organ—viz., that part which is supplied by the glosso-pharyngeal.

The seventh, auditory, is a nerve of special sense.

The eighth, glosso-pharyngeal, has a triple function, as shown in the table.

The ninth, or hypoglossal, is the principal motor nerve of the tongue.

CHAPTER III.

THE SYMPTOMATOLOGY OF BRAIN
DISEASE.

AN inquiry into the symptoms of brain disease necessarily presupposes a knowledge of the normal functions of the cerebral organ, as a perversion of these functions furnishes the principal means whereby departures from the healthy state can be detected. It is to be hoped, therefore, that the physiological facts already adduced will prove sufficient to meet the present requirements.

I propose first to review the leading features of cephalic semeiology, and then to discuss shortly the means employed in eliciting such signs as may require a certain amount of skill in their discovery.

MENTAL DISTURBANCES.

In brain disease the intellectual faculties may or may not be involved. It is seldom, however, that a careful observer will fail to discover some evidence of mental defect. The investigation, not always easy, must be prosecuted in a judicious way so as to avoid imparting to the patient, by exciting or otherwise influencing him, a mental condition which may be foreign to him when undisturbed. A careless mode of interrogating a patient when

his intellectual faculties are on trial will almost certainly fail to yield the required facts. I have frequently seen a patient who has been declared altogether free from mental disturbance by his attendant, "shown up" in that direction by a skilled practitioner of psychological medicine. I do not at all refer to the subtleties of mental aberration known only to the few, but to gross disturbances, such as loss of memory, emotional conditions, &c. The mental symptoms which present themselves are:—irritability of temper, absence of mind, emotional phenomena, fatuous manner, impaired memory, incoherence, hallucinations, melancholia, &c.

The facies, or facial appearance of a subject of cerebral disease is often very characteristic, frequently combining a fatuous, weak expression with an aspect of stolid indifference. But even in gross brain disease it may be impossible to detect the slightest mental aberration.

DISTURBANCES OF SPEECH.

The faculty of speech is often affected; such disturbance may have a paralytic or motor origin, as when the hypoglossal nerves are involved, which may occur in the medulla (nuclei), in the pons, or in the course of the fibres still higher up; the condition is then called anarthria, or dysarthria.

Again, language may be involved whilst the

lingual movements and the intellectual faculties are practically unimpaired, as in ataxic aphasia; this latter condition being the result of a lesion affecting the island of Reil and the adjacent part of the inferior frontal convolution, generally on the left side, and, in the great majority of cases, associated with right-sided hemiplegia. In other words, in this condition (ataxic aphasia) there is partial or complete impairment of the functions of that centre to which is ordinarily referred the co-ordination of impulses necessary to produce an intelligent expression of names or propositions in speaking and writing. Or a third case may occur in which a patient is unable to give a verbal expression to his ideas through the impairment of another centre, whose function is to endow the mind with the mnemonic faculty of association; and, therefore, the word with which he has in times past associated the external object fails him (amnesic aphasia). Amnesic aphasia is really the derangement of speech due to imperfection in the receptive or reproductive power of word-memory. Such impairment, according to Broadbent, may be found in a lesion of the supra-marginal and postero-parietal lobules, the angular gyrus, the posterior part of the infra-marginal convolution, and the convolutions bounding the parallel and collateral fissures. In ataxic aphasia the defect is more mechanical than mental, *i.e.*, the parts are no longer working in harmony; in amnesic aphasia

the defect is mnemonic, or more mental than mechanical. In reality, however, it is questionable whether cases of ataxic aphasia ever do occur without some mnemonic defect.

In addition to the varieties of speech-disturbance already named, there are other departures from the normal, such as the scanning or syllabic, or when the patient "clips" his words like a drunken man.

MOTOR DERANGEMENTS.

Derangement of the motor functions of the brain are evidenced in three ways:—by irritation, which is manifested by convulsions, tonic, clonic,* or choreic; in less severe cases by spasms or cramps of the muscles of the face or limbs; or by paralysis in its many forms, whether of a single ocular muscle alone, or of nearly all the voluntary muscles of the body. Hemiplegia is the commonest type of cerebral motor paralysis. It must, however, be recollected that paralysis of this form by no means involves the whole of one lateral half of the body, for the muscles of the back, neck, thorax, and abdomen, and those supplied by the upper filaments of the seventh escape; in other words, the muscles which are physiologically related on the two sides of the body are exempt in hemiplegia. Inco-ordination, an inability to co-ordinate

* *Tonic* spasms are continuous, and *Clonic* are intermitting.

the movements of the trunk and limbs, is most frequently observed in lesions of the cerebellum, pons, crura, and corpora quadrigemina, when the patient may be unable to walk or stand. Less frequently the movements of the limbs of one side are impaired in this direction, though cases do occur, and recently I recorded two, in which unilateral inco-ordination was the only result of a sudden brain seizure.

SENSORY DISTURBANCES.

Disorders of sensibility are either subjective or objective. The subjective disturbances consist of indefinite pains, formication, a sense of heaviness in the limbs, "needles and pins," &c. The objective signs show themselves in a variety of forms, tactile sensibility being generally impaired earliest, and subsequently, should the disease invade the sensory tracts more extensively, the conduction of pain and of all kinds of sensory impressions may be interrupted. We know that lesions affecting the radiating fibres behind the lenticular nucleus are especially prone to cause alterations of sensibility; but disease involving the sensory fibres in the external portion of the cerebral peduncle and in the pons and medulla will also cause sensory paralysis.

Disorders of special sense constitute symptoms of the greatest importance. The organ which furnishes the most valuable information is un-

doubtedly the eye. The other special senses—hearing, smell, taste, are also at times affected, especially when the base of the brain is the seat of the lesion.

As cerebral symptoms, headache and vomiting call for special notice. It may be accepted that the pain in the head which so frequently attends brain disease depends upon either increased intracranial tension, in which case the headache is the result of stretching of the dura mater or pia mater (there are anatomical reasons why the former should be the seat of the pain rather than the latter); or else it is due to inflammatory irritation, when the membranes need not necessarily be affected, for although the brain substance itself is insensitive, it is yet reasonable to assume, from facts relating to blood-vessels in other parts of the body, that under certain circumstances the vascular walls may become sensitive. The headache of cerebral tumour is no doubt the result, as a rule, of increased intracranial pressure; the more rapid the growth of the disease and the nearer to the surface it is situated, the more marked is the symptom. In some cases of rapid effusion into the ventricles without inflammation, the headache may arise from the same cause; indeed, in several brain affections attended by pain in the head the cause is increased intracranial pressure.

In inflammation of the brain, even though the

membranes are not implicated, there is generally, as I have already indicated, some complaint of pain. But when the membranes are affected, the symptom becomes a most prominent one, the excruciating character of the pain in acute meningitis being well known. Inflammatory lesions of the deep structures of the brain—basal ganglia, &c.—have been tolerably frequently met with unaccompanied by pain; as in tumour, the nearer to the cortical structures the lesion is situated the more prominent is the pain. In many cases the headache is localised, and coincides almost exactly with the seat of the lesion; in cerebellar tumours, for example, the pain is very often occipital. Although such is by no means a constant rule, it nevertheless so frequently happens, even in cases of softening from embolic attack, that the pain is referred to the region of the disease as to make it an important feature in the regional diagnosis. On the other hand, the pain may be general, or localised in a spot quite removed from the seat of a limited lesion. The attention of physicians has been directed lately to percussion of the cranium as a means of ascertaining the situation of a lesion. Dr. Ferrier, in an interesting article in *Brain*, quotes cases in which the complaint of pain has been elicited on tapping the cranial vault over the affected part of the brain. Personally, I have found the procedure useful.

Vomiting in cerebral disease is very often associated with headache; indeed, it is generally in such cases as tumour and meningitis, where headache is so frequently present, that we meet with vomiting. It not unfrequently happens that the vomiting is worst when the pain is at its height; though this is by no means a rule, for occasionally the headache will alternate with the vomiting. The vomiting is most probably reflex, and is to be accounted for by the close connection between the nucleus of the vagus and the long or inferior nucleus of the fifth in the medulla; any sensory nerves that exist in the dura mater being derived from the trigeminus.

CHAPTER IV.

METHODS OF INVESTIGATING THE SYMPTOMS OF BRAIN DISEASE.

It is always important to make special inquiry from the friends of a patient regarding his mental faculties, as little points may easily be overlooked unless we have before us such details as can only be furnished by an observant friend. Again, we sometimes discover on inquiry that a peculiarity in manner shown by a patient is perfectly natural to him.

Extensive paralysis can generally be recognised without difficulty. The observer should in every case ascertain exactly for himself the degree to which mobility has become impaired, as the statement of the patient or his friends is occasionally very misleading. For example, a patient with a cerebellar tumour will not unfrequently complain that "he has lost the use of his legs," the fact being that the motor impairment is rather an inco-ordination than a paralysis, so that, although he cannot walk, he can display vigorous action when lying in bed. It must not be forgotten that the paralysis may be extremely limited in its extent; it may be confined to a single muscle, or to a group of muscles. When limited to a few muscles, and only

partial (incomplete), it is often very difficult to detect. In every case of suspected cerebral disease, the facial, lingual, and ocular muscles should be very carefully investigated. In the case of the facial muscles a slight paralysis may be discovered by directing the patient to display his teeth, when the mouth will be found to be drawn to the healthy side. In hypoglossal paralysis the patient will protrude his tongue towards the paralysed side; the protrusion should be made energetically, and care taken to observe that the deviation is real, and not only apparent from dragging of the mouth to one side. It is not sufficient to direct a patient to protrude his tongue, but his power of turning the tip upwards, both within and outside the mouth, should also be tested.

A few words are necessary regarding the paralyses of the muscles of the eye.

When the external rectus is paralysed, the fact may be detected by holding an object in front, a few feet distant; both eyes are then fixed upon it, and it is only when the object is made to move towards the affected eye that the want of action in the muscle becomes apparent; the optic axes then cease to work together, and double vision (diplopia) is the result. The diplopia will be found to be direct in this case—that is, the image, as seen by the paralysed eye, is projected towards the temple of the same side. In cases of old-

standing paralysis of the external rectus there may be a marked internal squint from contraction of the opposing muscle; this convergent strabismus will be made very apparent if an object be held before the patient's face and the sound eye closed, when the squinting one will turn slowly out and become fixed upon the object, but it again rolls inwards when the other eye is opened.

Paralysis of all the muscles supplied by the third nerve is easily recognised by ptosis (inability to raise the upper eyelid); external squint, or, more correctly speaking, a limitation of the movements of the eye to the outward direction; and sluggish and somewhat dilated pupil. Diplopia will be observed when an object is held above, below, or at the side opposite to the affected eye. Not unfrequently the lesion is limited to a few of the fibres of the third nerve. In paralysis of the internal rectus the eye cannot be moved inwards, and a crossed diplopia results if an object be held towards the side of the sound eye; that is, the object as seen by the paralysed eye is projected inwards. This double vision is most apparent when the object is held not only towards the opposite side but above the level of the eye.

Paralysis of the superior rectus gives rise to a limitation of the movements of the eye above the horizontal meridian plane. When an object is moved from below upwards above this level the

eye cannot follow it. The reverse obtains in cases of paralysis of the inferior rectus.

A patient suffering from paralysis of the superior oblique generally complains of double vision when an object is held below the level of the eye. The false image will be found to be below and to the outer side of the real one; it also appears to be nearer to the patient than it really is.

It is scarcely necessary to remind the reader that a large number of the cases of squint are not due to paralysis of the nerves of the eye, but to other causes—errors of refraction, &c.

Attention to the following point will materially aid in drawing a distinction between the two classes of cases:—in paralytic squint, if an object be held towards the weak side of the affected eye (for example, to the left of the left eye in paralysis of the external rectus), the sound eye being closed, the affected one will slowly rotate towards the object, and at the same time the closed eye will also rotate, but to a greater extent. This obtains only in cases of squint having a paralytic origin.

The great importance of attending to the behaviour of paralysed muscles when faradised and galvanised is now admitted by all interested in the diagnosis of nervous affections. In practising electro-diagnosis both faradic and galvanic currents must be employed. A muscle may be made to contract either by *direct or indirect* stimulation:

54 *Employment of Electricity in Diagnosis.*

if the former, the muscle itself is acted upon; if the latter, the motor nerve supplying the muscle is stimulated.

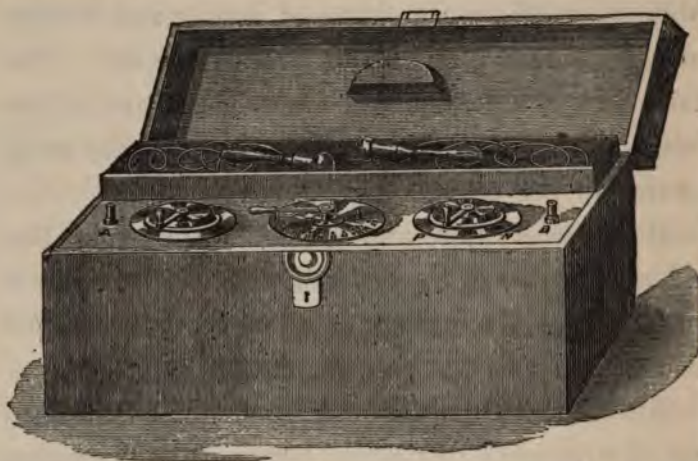


FIG. 20.—*A very convenient form of Galvanic Battery, 40 Cells Leclanché.*

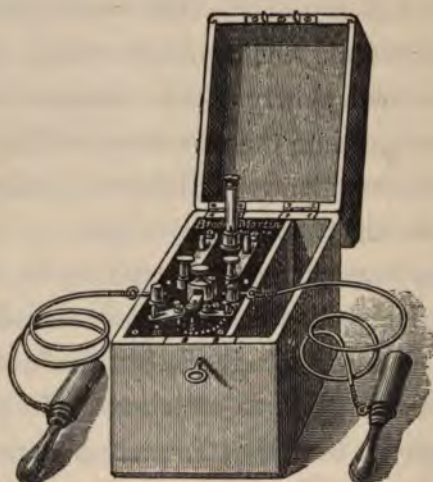


FIG. 21.—*An exceedingly cheap and convenient Faradic Battery by Messrs. Brady & Martin.*

In the following brief description of the electrical features of motor paralysis I prefer to retain the German symbols. The positive pole, or anode, is represented by A; the negative, or kathode, by K; closing of the circuit, by S (*Schliessung*, shutting); breaking or opening of the current, by O (*Oeffnung*, opening); contraction by Z (*Zuckung*, contraction). Z generally stands for strong contraction, and z for a weak contraction. A strong contraction in a muscle taking place on kathodal closing is represented by KSZ; and so forth. Muscles in a normal condition will contract to both forms of current, and the same amount of contraction will be induced whether the current be applied to the motor nerve or to the muscle itself.

Muscular contractions occur at the closing (S) and at the opening (O) of a galvanic current. The weakest current which will induce a contraction does so at kathodal closing (KS)—*i.e.*, the negative pole placed upon the muscle. With a stronger (medium) current the kathode causes stronger contractions on closure of the circuit, but not on breaking; whilst with the same current the anode causes feeble contractions on the opening and closing of the circuit. Thus, with a medium current we have in the normal condition KSZ, ASz, and AOz. With a strong current contractions occur with the kathode and anode at both the make and the break.

Abnormal changes may be quantitative, or qualitative, or both: that is, the law of muscular contraction remaining unaltered, the amount of contraction induced by a current of a certain strength may be lessened—quantitative; or the change may consist in lost faradic with increased galvanic excitability; or the anodal closing contraction may become stronger than the kathodal—qualitative.

The quantitative and qualitative changes that are observed as departures from health when muscles are tested by faradic and galvanic currents enable us in many cases to form an opinion as to the situation of the lesion. In certain forms of spinal and peripheral paralysis these abnormal changes are very marked, constituting what is known as Erb's *reaction of degeneration*. In this state the change is first quantitative, the excitability of the muscles to the faradic currents is early lost, the galvanic excitability declining also during the first few days. But after the lapse of a week or so the galvanic excitability increases; so that a weak current, hitherto insufficient to cause contraction, will affect the muscles distinctly. Along with this quantitative change qualitative changes make their appearance, the contractions induced are sluggish, and the anodal closing contraction, which we saw was feebler in health than the kathodal closing, may become stronger than the

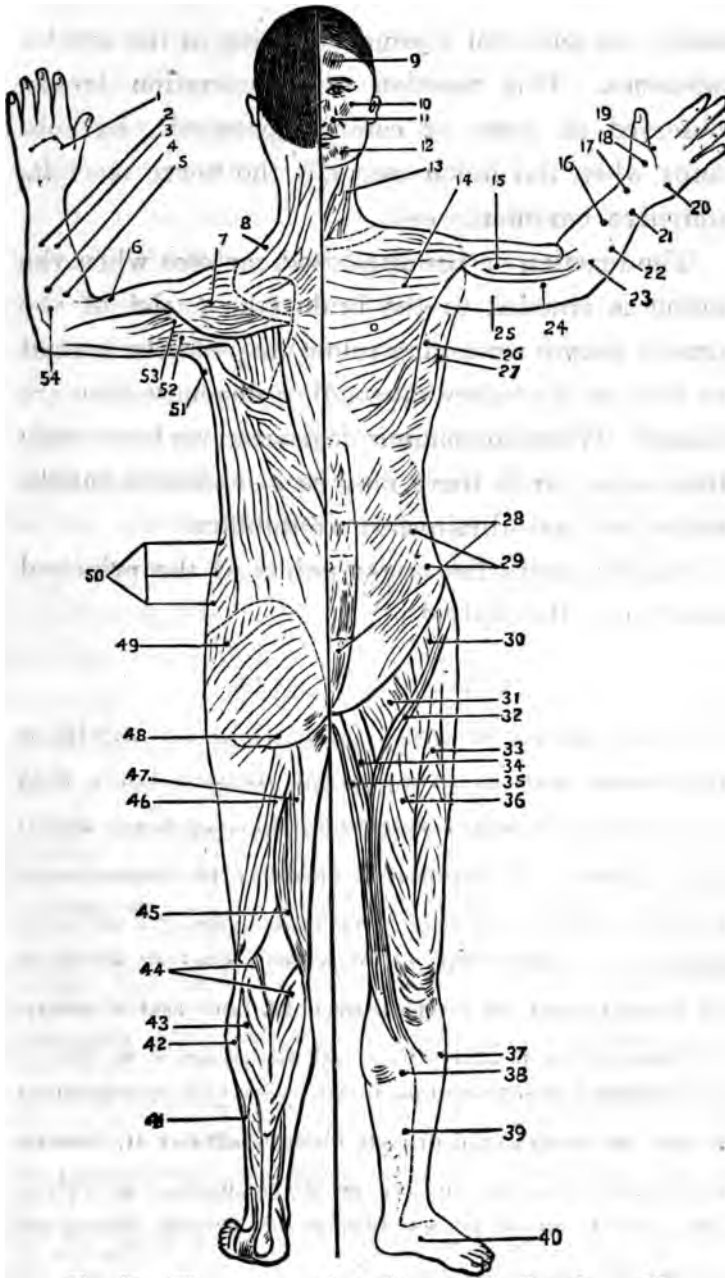


FIG. 22.—The numbers refer to the note at the foot of page 68.

latter, the kathodal closing declining as the anodal advances. This reaction of degeneration is not observed in cases of cerebral paralysis, but obtains when the lesion cuts off the nerve from its nutritive ganglionic cells.

The reaction of the paralysed muscles when the lesion is situated in the brain (the nuclei of the cranial nerves remaining intact) is perfectly normal so long as secondary descending degenerations are absent. When descending degenerations have made their appearance there may be a moderate quantitative decline—diminished excitability.

Fig. 22 shows the motor points of the principal muscles of the body.*

CONVULSIVE ATTACKS.

It is a matter of great moment to distinguish a convulsion due to gross brain disease from that

* 1. Interossei (dorsal): 2. Extensor indicis: 3. Extensors of thumb: 4. Extensors of fingers: 5. Extensor carp. rad. brev.: 6. Supinator long.: 7. Deltoid: 8. Trapezius: 9. Frontalis: 10. Zygomatici: 11. Muscles of upper lip: 12. Muscles of lower lip: 13. Sterno-mastoid: 14. Pectoralis major: 15. Biceps: 16. Flexor carpi rad.: 17. Flexor long. Pol.: 18. Opponens Pol.: 19. Adductor Pol.: 20. Flexors of little fingers: 21. Flexor subl. digit.: 22. Flexor carpi ulnaris: 23. Pronator teres: 24. Brachialis ant.: 25. Inner head of triceps: 26. Serratus mag.: 27. Latissimus dorsi: 28. Rectus abdominis: 29. External and internal oblique, and transversalis: 30. Iliacus: 31. Pectineus: 32. Sartorius: 33. Rectus femoris: 34. Adductor long.: 35. Adductor mag.: 36. Vastus internus: 37. Tibialis anticus: 38. Soleus: 39. Flexor long. digit.: 40. Abductor hallucis: 41. Peroneus brevis: 42. Peroneus longus: 43. Soleus: 44. Gastrocnemius: 45. Semimembranosus: 46. Biceps: 47. Semitendinosus: 48. Gluteus Max.: 49. Gluteus med.: 50. External and internal oblique, and transversalis: 51. Latissimus dorsi: 52. Outer, 53. Middle, head of triceps: 54. Extensor carpi uln.

which is hysterical or epileptic in origin. The organic convulsion very frequently begins locally, and may be confined to one side; this is especially true of tumours of the brain. However, a unilateral commencement is frequently met with in functional epilepsy, and, on the other hand, the convulsions of organic cerebral disease may not commence locally, and may present the commonest of epileptic auræ. A local commencement should excite the suspicion of organic disease, and this suspicion would be strengthened by the limitation of the seizure to certain groups of muscles. Care must be exercised in distinguishing between cases of unilateral functional epilepsy followed by "post-epileptic paralysis" of the hemiplegic type, and cases of acute cerebral lesion—hæmorrhage or embolism—attended by convulsions. In the former the paralysis will be transient and incomplete; but when the paralysis and convulsions are due to an acute organic lesion the paralysis is generally more pronounced and lasting. Well-marked transient paralysis (sometimes lasting a few days) following a convulsion and affecting the convulsed parts, is often observed in cases of chronic cerebral disease. In attempting to make out the cause of a convulsion the character of the attack must be considered along with other symptoms of organic disease—headache, vomiting, double optic neuritis, implication of cranial nerves, &c. The headache of

functional epilepsy generally passes away soon after the fit; but in organic disease it will remain during the intervals.*

SPECIAL SENSES.

The olfactory nerves (sense of smell) may be altered in a hyperæsthetic direction (hyperosmia); or the sense may be diminished or lost (anosmia). The latter condition occurs more frequently and is more important than the former. In testing this sense the condition of the nasal mucous membrane should be noted, as changes in it—coryza, &c.—will abolish smell. The substances employed should be varied and of such a character as will enable the observer to draw a distinction between the olfactory nerves and the nerves of common sensation (the 5th), for when the latter are paralysed the membrane loses its reflex excitability. Irritating substances, such as smelling-salts, bromine, &c., affect the fifth nerve, but are not appreciated as true olfactory impressions. In hysterical cases, for example, it is common enough to find that odoriferous particles, sweet-smelling flowers, &c., can be recognised when the strongest smelling-salts produce no effect.

The gustatory sense (taste) is difficult to test with accuracy. The glosso-pharyngeal presides exclusively over the sense of bitterness, and, as

* For further particulars, see Diagnosis of Epilepsy.

the nerve has a definite distribution (to the posterior third of the tongue), the fact becomes an important one. The other gustatory perceptions appertain to the functions of the gustatory division of the fifth and the chorda tympani, probably almost exclusively to the latter. The sense of taste may be more active than usual (hypergeusia), or it may be lessened or abolished (ageusia). Exaggeration of the gustatory sense is sometimes observed in hysterical conditions and also in some forms of melancholia. To test taste the substances employed should be rubbed into the tongue when it is protruded, and the patient directed to form an opinion before the tongue is withdrawn.

The auditory sense (hearing) may be tested by the tuning-fork. When the disorder is a nerve deafness no sound is heard in the affected ear when the meatus is closed and the sounding-fork applied to the skull; whereas in health the tuning-fork when thus placed sounds louder in the closed ear than the open one. Or Brenner's galvanic method may be employed. In the normal condition sounds are heard when one pole is placed on the back of the neck and the other on the external auditory meatus under the following conditions, viz.—at the closure of the kathode (negative pole on the ear); during the passage of the current, the negative pole still on the ear; and at the opening of the anode (the positive pole on the ear).

Departures from these normal reactions are detected when the portio mollis is hyperæsthetic, or depressed. When the sense is exalted quantitative changes are noted—*i.e.*, loud ringing is heard on using a very weak current at kathodal closing, and persisting until the current is broken. This quantitative change is less marked at anodal opening. In more exaggerated conditions qualitative changes may be present—*e.g.*, hissing sound at kathodal opening and anodal closing.

In auditory anæsthesia the normal reactions are either absent or badly marked.

The optic apparatus should be most carefully investigated in all cases of suspected brain disease. No one can hope to make progress in the diagnosis of cerebral disease without some knowledge of the ophthalmoscope; indeed, it is a *sine quâ non* in nervous diseases, and may be said to be of as much importance in brain disease as the stethoscope is in thoracic disorders. As space is necessarily too limited to permit me to enter at all fully upon the various phases of visual disturbance, as met with in cerebral disease,—hemiopia, diplopia, amblyopia, amaurosis, &c., I shall simply make a few remarks upon the most important change which can be detected by the ophthalmoscope, namely, optic neuritis. The pupil should be well dilated with atropine before a positive opinion is formed as to the condition of the disc and retinal vessels. It

must never be forgotten that the patient may see perfectly well whilst his discs are swollen and almost obliterated; so that we may meet with the most intense double optic neuritis—swollen discs, large veins, hæmorrhages, vessels appearing and disappearing amongst the œdematous effusion in the fundus, &c., and perfect vision. It is true that swelling of the disc occurs in other affections besides intracranial lesions; for example, in granular kidney, in lead poisoning, and intense anæmia. But taken along with such symptoms as headache, vomiting, cranial nerve paralysis, especially if there be no albuminuria or history of lead, the condition conclusively points to organic brain disease—generally tumour—though it is often found in meningitis and other cerebral affections (*see Frontispiece*).

CHAPTER V.

APOPLEXY

(Cerebral Hæmorrhage).

By cerebral hæmorrhage is meant the effusion of blood into the substance of the brain or its ventricles.

MORBID ANATOMY.

Cerebral hæmorrhages vary very considerably in extent. They occur either as capillary extravasations, usually found in the cortical gray matter, or as isolated effusions, some of which are exceedingly large, and may occupy the greater part of one hemisphere: in some cases as much as eight or ten ounces of blood are poured out. As a rule these hæmorrhages vary in size from a small hazel nut to a large walnut. When fresh the apoplectic clot is generally dark, and towards its periphery more or less mixed with broken-up brain matter. The limiting wall consists of brain *débris*, outside which lies a zone of softened tissue infiltrated with blood serum, and this zone is often the seat of capillary hæmorrhages. When recovery takes place the effused blood and broken-up brain matter gradually disappear, and in their place may be found, a few months after

the attack, either a cyst containing yellow or brown fluid and some loose spongy connective tissue, or else cicatricial tissue which may have been preceded by a cyst.

ETIOLOGY.

Though cerebral hæmorrhage occurs most frequently in advanced life, and seldom below the age of forty, it must be recollected that early life by no means precludes the occurrence of such a lesion. According to Gintrac's statistics the decade from sixty to seventy showed the greatest proportion of cases. In childhood meningeal hæmorrhages are proportionately much more frequent than in advanced age. Men are more frequently attacked than women, being more exposed to the influences which predispose to the affection.

The most important predisposing cause exists in the degenerations to which the arteries of the brain are liable. In the majority of cases small miliary aneurisms are found situated upon the degenerated vessels. The form of degeneration which leads to the formation of these small aneurisms, the significance of which was correctly interpreted by Charcot and Bouchard, is said to be a kind of arterial sclerosis. The rupture may take place as the result of weakness of the vessel from a fatty degeneration without aneurismal

dilation, a fact to which Paget called attention thirty years ago. The atheromatous rigidity which so frequently affects the basal vessels, probably aids in the production of the intra-cerebral miliary aneurisms by interfering with the modifying influence which the elastic walls usually exert upon the pulse wave. The arterial sclerosis and accompanying aneurismal dilatations are most frequently found, and in a more pronounced degree, in and in the neighbourhood of the caudate and lenticular nuclei of the corpus striatum, and after these parts in the optic thalami; in other words, the ramifications of the middle cerebral arteries are most frequently diseased. When once the vascular degeneration has become established, the risk of rupture is always more or less considerable; but the actual laceration is probably in most cases preceded by an increase in the cerebral blood-pressure. The causes which give rise to an increase in the vascular pressure are very numerous, and may exist in the brain itself, or be dependent on the operation of some distant influences. Epileptic fits, violent emotional displays, passive congestions following thrombosis of the sinuses or of the veins of the pia mater, &c., will elevate the blood pressure in the cerebral vessels. Of the distant causes which tend to increase the arterial tension, the following may be mentioned:—hypertrophy of the left ventricle; the contracted kidney and general arterio-

capillary fibrosis (?) which so frequently accompany the cardiac hypertrophy; exposure to cold, so that the superficial arterioles become contracted, and thus the blood pressure accordingly raised.

SYMPTOMS.

The semeiology of cerebral hæmorrhage may very conveniently be divided into the premonitory symptoms, the features which distinguish the onset, and the subsequent evidences of cerebral disease,—the sequences of cerebral apoplexy.

Premonitory symptoms.—The prodromal symptoms which often precede the vascular rupture are indicative of cerebral irritation, and commonly arise from disturbances of the intra-cranial circulation, which not unfrequently amount to minute hæmorrhages. Writers on the subject have described a great variety of symptoms of this class, among which the most important are the following :—transient aphasic attacks, frequently recurring dizziness, singing in the ears, headache, neuralgic pains, attacks of motor weakness in the parts subsequently paralysed, subjective sensory phenomena — “needles and pins,” &c. These warnings may show themselves days, it may be weeks, before the onset of the attack. Whilst due stress is laid upon the premonitory symptoms, it must not be lost sight of that in many cases of cerebral hæmorrhage they are entirely absent.

The onset.—The mode of attack varies considerably. In the majority of cases the symptoms are undoubtedly of an apoplectic character (limiting the term apoplectic entirely to attacks attended by loss of consciousness). In some the loss of consciousness is sudden and complete, in others it is only manifested after a stage of mental confusion. In a considerable number of cases, however, the onset is not accompanied by any loss of consciousness. In the usual apoplectic attack it generally happens that the patient, immediately before losing consciousness, complains of some of the prodromata—headache, dizziness, sleepiness, inability to speak, &c.; and this pre-apoplectic stage may, and often does, last for some hours before the stage of insensibility comes on. Occasionally loss of power over an extremity supervenes before consciousness is lost. When the coma is profound reflex excitability is generally abolished, with the exception of the pharyngeal reflex—swallowing—which commonly persists; respiration is irregular and laboured, the cheeks being puffed out in expiration; the face is flushed and tumefied. If paralysis be present the hemiplegia is shown by the utter helplessness discoverable in the limbs of one side, which when raised drop in a lifeless manner, and the corner of the mouth will be observed to occupy a lower position on the affected side. The temperature is lowered, but subsequently rises to,

or slightly above, the normal, as consciousness returns. Conjugate deviation of the eyes, with rotation of the head towards the non-paralysed,—*i.e.*, the diseased side of the brain—very often occurs. The gradual return of the reflexes indicates the approaching restoration to consciousness.

Another mode of onset, the epileptiform, should be mentioned, which is closely allied to the apoplectiform just described. When the seizure is of this nature the patient drops down convulsed as well as insensible, and will commonly be found paralysed on one side. Usually the paralysis attacks the convulsed side, should the convulsions be unilateral. At times the convulsions continue after the paralysis has appeared, in which case it not unfrequently happens that the sound side is alone affected with the clonic spasms,—a fact which is generally taken to be indicative of damage to the opposite cerebral hemisphere as well.

As already referred to, a considerable number of the cases of cerebral hæmorrhage present quite a different onset. Consciousness may be retained, and the patient, having experienced little else than a momentary giddiness, is made aware of the powerless condition of the affected limb or limbs by a numb feeling in them. This mode of attack, which Bastian terms the “simple onset,” is very frequent in the slighter forms of hemiplegia from hæmorrhage.

THE SEQUENCES OF CEREBRAL HÆMORRHAGE.

Following the period of onset is often a stage of *inflammatory reaction*, which may occasion considerable anxiety to the physician. In the majority of cases the inflammatory symptoms appear a few days after the attack. At this stage headache often supervenes, the temperature may be considerably elevated, delirium is not uncommon, and *early rigidity* may develop.

When the acute symptoms have passed away the chronic aspect of the affection manifests itself. With rare exceptions the paralysis is hemiplegic, and affects the side of the body opposite to that on which the cerebral lesion occurred.

The typical hemiplegia (hæmorrhage into, or just outside the corpus striatum) occurs in the form of paralysis of the arm and leg, the lower muscles of the face, and one side of the tongue—all on the side opposite to the brain lesion.

Disturbances of sensibility are often noted together with the motor paralysis, but the impairment is seldom extreme, and sensation very frequently returns about seven or ten days after the onset, leaving, however, subjective sensations. The continuance of marked anæsthesia furnishes an important clue to the regional diagnosis. The speech is frequently “thick” at first, but in hopeful cases, provided the speech centre is not damaged and the lingual movements are not materially affected,

this difficulty in articulation soon passes off. The tongue generally recovers early. The paralysis of the face begins to disappear after a week or so. The last sign to disappear is the paralysis of the limbs, and the limb most paralysed—as a rule the arm—recovers last. It very often happens that the leg has become a tolerably useful limb whilst the arm is still more or less helpless.

The electrical irritability of the paralysed muscles may be somewhat increased during the first few days when tested by both kinds of current; as a rule, however, it is normal, and continues so unless secondary descending degeneration of the motor tract occurs, or wasting from disuse supervenes, when it may become slightly diminished.

The skin reflexes (plantar, cremasteric, abdominal, &c.) are often increased on both sides, more especially on the sound one, but they may be quite abolished. The tendon-jerk phenomenon (ankle clonus, knee-jerk, &c.) is nearly always exalted on the paralysed side, and that even before the development of descending degenerative changes.

Contractures (rigidity) are of frequent occurrence. The condition is seen more frequently in the arm than in the leg. Occasionally the spasticity of the muscles shows itself at the time of the hæmorrhage. According to Durand-Fardel this occurs with special frequency when the hæmorrhage

has destroyed the walls of the lateral ventricles. The spasmodic condition which attends such an event always disappears in a few days, should the patient survive so long.

Again, rigidity may occur at the period of *inflammatory reaction*, when it constitutes the so-called “early rigidity” of Todd. According to Dr. Todd the inflammatory condition is entirely responsible for the occurrence of contractures at this stage. The rigidity may be limited to the fingers of the paralysed upper extremity, or the arm may be bent, and the wrist and fingers also flexed. In some cases the leg is affected as well, when the thigh will be found drawn up, and the knee firmly flexed. The spasm may pass off soon, or it may persist for months.

The commonest form of rigidity is that which occurs in the later stages of the disease,—the so-called “late rigidity” of Todd. It is probable that in this case the contractures are due to descending degeneration of the fibres of the pyramidal tract of the opposite lateral column of the cord. This view is, however, denied by Hitzig, who ascribes the condition to involuntary associated movements in the paralysed muscles—movements initiated in the cortex of the sound side, but which, by irradiation, have found their way into the channels the upper tracts or centres of which have been destroyed.

Erb has not observed "late rigidity" before the second month of the affection. Though its occurrence is often postponed to even a very much later date, it can scarcely be doubted that the spastic condition occasionally observed two or three weeks after the hæmorrhage is due to a precisely similar agency, and therefore may properly be termed "late rigidity." As in the case of "early rigidity," the spasm may be limited to the flexors of the fingers, but the forearm and wrist are often flexed as well. When the leg is affected, extension predominates at first, but subsequently, after the lapse of months or years, contraction of the hamstring muscles may occur. In some cases the paralysed facial muscles become affected by a similar spasticity. Forcible attempts to straighten the flexed limbs generally produce considerable pain.

The paralysed muscles may be affected, but rarely, by tremor resembling paralysis agitans, or by choreic movements. In a case of right hemiplegia with paralysis of the left third nerve (crus lesion) at present under my care, the patient is the subject of right hemichorea.

The mental disturbances which are prone to follow cerebral hæmorrhage are loss of memory, more especially for recent events, and attacks of emotional weakness; as a rule, patients so affected never quite regain their mental calibre.

Bed-sores affecting the paralysed side occasion-

ally develop soon after an extensive extravasation; in the majority of cases they are met with in the middle of one of the nates. Œdema of the paralysed extremities is of common occurrence a few hours after the onset, and may last for a long time. The joints on the affected side sometimes become inflamed, a complication which usually occurs about the second or third week after the cerebral hæmorrhage. These changes may with truth be ascribed to an interference with the trophic and vaso-motor nerves.

DIAGNOSIS.

It will be convenient to refer to the diagnostic questions as they present themselves for solution in a chronological order. To begin with, is the state of coma or stupor in which the patient has been found due to vascular changes—hæmorrhage, embolism, or thrombosis? or has the condition arisen from other causes—uræmia, alcoholic intoxication, injury to the head, narcotic poisoning, or an epileptic fit? In distinguishing between *uræmic coma*, and an apoplectic condition due to hæmorrhage or softening (embolic attack), the age is of importance. Whilst recollecting that hæmorrhage seldom occurs below forty, it must not be forgotten that occlusion of a vessel is often met with at an earlier period; and further, that cerebral hæmorrhage very often complicates an advanced

renal case, so that the discovery of Bright's disease by no means renders the diagnosis of uræmia certain. The onset should be attended to. *Uræmic coma* almost always commences with convulsions, and is seldom profound. Again, in uræmia the temperature begins to fall with the occurrence of the coma, and continues to sink as long as the condition lasts, so that it may fall 6° or 8° F. below the normal in very severe cases.

The difficulties in the way of drawing a distinction between alcoholic intoxication and apoplexy are very great indeed. It often happens that the degenerated vessel ruptures under the influence of the increased strain induced by the unduly stimulated circulation; so that the detection of a vinous odour in the breath of the patient does not warrant the conclusion that he is "only drunk." Evidences of the cerebral lesion should be sought for, such as conjugate deviation of the eyes, elevation of one angle of the mouth, spasm confined to one side, utterly helpless condition of the limbs of one side, &c. Even to the skilful observer, fully alive to the necessity for caution, it often becomes a very difficult matter to decide between the two conditions.

Regarding the features which would aid in drawing a distinction between injury to the head and apoplectic coma there is little to be said. The signs of external injury should be sought for, but

even if found, it has yet to be determined whether a fit preceded the fall—presuming such to be the cause of the wound, or whether the coma is entirely the result of the accident.

Some cases of narcotic poisoning are with difficulty distinguished from the apoplectic state due to hæmorrhage. The bottle which contained the poison may furnish a clue, and the patient's breath may also yield information, as the odour of some of the narcotic poisons is distinctive. Opium-poisoning resembles hæmorrhage into the central portion of the pons very closely; in both cases the coma is most profound and the pupils markedly contracted. As Bastian points out, the onset of the opium-coma is slow, but in cases of hæmorrhage into the pons it is most sudden. Again, occasionally it happens that convulsions are associated with the pons lesion, whilst such is never the case in opium-poisoning.

The *epileptic coma* is practically indistinguishable from the *apoplectic*. The fact that the patient has been subject to epileptic fits would be presumptive evidence in favour of epilepsy, but it would by no means exclude apoplexy.

DIFFERENTIAL DIAGNOSIS OF HÆMORRHAGE FROM EMBOLISM AND THROMBOSIS.

It will often be impossible to distinguish between the above conditions; indeed, in the

majority of cases of apoplexy the precise nature of the lesion can be little more than conjectured. But there are certain indications which point to hæmorrhage in contradistinction to embolism and thrombosis; and under certain conditions an almost unreserved diagnosis may be made. A sudden onset preceded by prodromata occurring in old people points to hæmorrhage; a gradual one preceded by prodromata in old people points to thrombosis; a sudden one without any prodromata in young people points to embolism.

A very profound and lasting coma is indicative of hæmorrhage, whilst a rapid recovery from a well-marked hemiplegia is decidedly against that condition. In some embolic attacks, especially when a large number of small vessels are suddenly occluded, as in multiple embolism, the coma is also profound and lasting. In hæmorrhage a distinct remission of the symptoms, such as is occasionally to be met with about the third or fourth day in embolism, is seldom to be observed; but, on the contrary, slight relapses from inflammatory reaction are not unusual. The temperature will sometimes throw light upon the nature of the attack. In hæmorrhage there is often a period at the commencement in which the temperature is lowered, whilst this initial sinking is generally absent in embolism. Again, after the "initial lowering" of a hæmorrhage has passed off, the thermometer very

frequently indicates a rise of three or four degrees above the normal, the elevation as a rule lasting for some time; in embolism, on the other hand, it is not unusual to meet with a sudden rise of three or four degrees, or more, soon after the onset, followed speedily by a marked fall.

PROGNOSIS.

Cerebral hæmorrhage must always be considered a most serious affection. In all probability the attack will prove fatal, and that speedily, when the following symptoms are present:—very profound and lasting coma, running beyond twenty-four hours, well-marked stertor, complete relaxation of the sphincters, irregular pulse, slow and laboured respiration, very marked and persistent depression of temperature. Such cases result from large hæmorrhages into the centre of the hemisphere, often rupturing the walls of the lateral ventricles, from bleeding into the pons, or from large meningeal hæmorrhages.

During the next few days the occurrence of bed-sores, a sudden and continuous elevation of the temperature, and great difficulty in swallowing, should certainly excite alarm. A fresh hæmorrhage may be recognised by a sudden fall in the temperature, and a return of the coma. With the end of the second week will generally pass away

all risk of a fatal result as the immediate effect of the hæmorrhage itself.

The motor paralysis may be expected to commence to disappear, even in severe cases, by the end of the first week or ten days. Should a month elapse without some evidence of improvement in the lower extremity, the chances of anything like complete motor recovery are very scant indeed. The development of "early rigidity" must be regarded, so far as the subsequent recovery of motor power is concerned, as an unfortunate complication. There is no relation between the recovery of the leg and the arm beyond the fact that the former generally recovers first; for a patient may be able to walk considerable distances whilst his arm is still helpless. Occasionally the arm recovers first, an event which is generally regarded as presaging an evil result.

TREATMENT.

The therapeutical measures adopted are, first, such as are calculated to prevent an attack—prophylaxis; second, such as are directed against the attack itself; third, those that are appropriate for the case during the few days succeeding the attack; and fourth, those adopted with a view to relieve the results of the lesion.

Our prophylactic treatment can scarcely be expected to do more than counteract the exciting

causes of the extravasation, as until miliary aneurisms are formed, and prodromata reveal themselves in consequence, the existence of the vascular degeneration which is responsible for the rupture cannot even be suspected. As a rule, treatment intended to avert a threatened attack is only employed after the patient has already experienced a seizure, the object being to avoid the recurrence of the hæmorrhage.

Familiarity with the exciting and predisposing causes of cerebral hæmorrhage will enable us to suggest measures of general treatment which will aid in removing or preventing the occurrence of these causes; but each case will require special consideration in the light of the details it furnishes.

In treating the attack itself care must be taken lest an error be made in the direction of over-activity. Speaking generally, the days of drastic purgatives, bleeding, &c., are past. But whilst care must be taken not to do harm by too active interference, occasionally cases may arise in which it is judicious to abstract some blood by venesection; as, for example, when the seizure is attended by marked turgidity of the vessels of the face and neck, with a regular and powerfully-beating heart and pulsating carotids, when the patient is young and robust, and, in short, when the phenomena of cerebral hyperæmia are plainly present. Should the patient be able to swallow, such drugs may be

administered (but with great caution) as are calculated to counteract alarming action of the heart when it prevails, either in the direction of excessive force and rapidity, or irregularity and feebleness. In the former case bromide of potassium and aconite, in the latter ammonia or alcohol, might prove useful.

When the attack is slight it is unnecessary to do anything more than keep the room well ventilated and cool, raise the patient's head and apply cold, administer purgative enemata when necessary, and attend to the bladder.

Symptoms of irritation during the stage of inflammatory reaction must be combated by local bleeding—leeches applied behind the ear, cold to the head, attention to the bowels, and the exhibition of bromide of potassium; or, when there is much excitement and wakefulness, opium in small doses.

In conducting the case through the chronic phases of the affection the general health of the patient must be attended to in the most assiduous manner; indeed, beyond the influence exerted by general good health upon the absorption of the clot, it is highly probable that our therapeutical endeavours utterly fail in promoting that process. The practice of indiscriminately ordering so-called absorbents, with the object of hastening the absorption of the extravasated blood, is certainly to be deprecated. The writer has seen undoubted

harm done by the use of iodide of potassium and mercury. Tonics such as iron and quinine may be employed with advantage; or the mineral acids and bitter infusions. Some cases appear to improve during the administration of such remedies as the tribasic phosphate of silver in doses of one quarter of a grain, or the phosphide of zinc in doses of the eighth or tenth of a grain.

Electricity should not be employed too early; but after the lapse of a few weeks, should the muscles remain paralysed and flaccid, the interrupted current does good. It is useful to faradise the extensor muscles when a limb has become contracted; or the constant current may be applied to the rigid muscles (the flexors) with, at times, considerable relief. Friction of the skin, with or without a stimulating liniment, and passive movements of the paralysed limbs, should not be neglected.

CHAPTER VI.

MENINGEAL HÆMORRHAGE.

It is convenient here to describe extravasation of blood upon the surface of the brain, as meningeal and intra-cerebral hæmorrhage are in many respects closely allied.

MORBID ANATOMY.

Occasionally the clot forms between the dura mater and the skull; though generally the hæmorrhage takes place between the dura mater and the arachnoid. The blood may be effused into the sub-arachnoid space, but, clinically speaking, it is impossible to distinguish between effusions above and beneath the arachnoid. When the arachnoid space is the seat of the hæmorrhage, either as the result of laceration of the dura mater or of the blood finding its way from the pia, the clot may be so extensive as to cover an entire hemisphere.

According to Virchow, the hæmorrhage takes place into the arachnoid space in cases of meningeal apoplexy of young children.

The appearances presented by the brain vary very much. Hæmorrhages beneath the dura mater, if large, as they often are, flatten the convolutions, but seldom lacerate the brain substance; but when

the effusion proceeds from a large vessel at the base there may be very considerable destruction of cerebral tissue.

The quantity of effused blood varies from a clot the size of a pea to a large mass equal to half the size of the hemisphere. It is probable that small hæmorrhages may in time be absorbed, but large effusions invariably prove fatal. Occasionally the blood finds its way into the lateral ventricles, and sometimes even into the fourth ventricle and central canal of the cord.

ETIOLOGY.

Most of the cases arise from injuries to the skull, arteries, sinuses, or pia-matral capillaries being lacerated in consequence. Aneurisms situated upon the arteries at the base of the brain may rupture and give rise to the effusion. The middle cerebral is most frequently affected with aneurism, and next probably the basilar artery. Rupture may occur in the veins from stasis due to thrombosis of the sinuses. In the course of acute infectious diseases meningeal hæmorrhage may arise; also as a result of the degenerations which occur in general paralysis of the insane. In some forceps cases, or in prolonged and difficult births without instrumental aid, laceration of the membranes and consequent hæmorrhage may occur, constituting the meningeal apoplexy of new-born children.

SYMPTOMS AND DIAGNOSIS.

The cases which owe their origin to injury to the skull are very difficult to diagnose, as it is almost impossible to separate the symptoms due to the concussion from those dependent upon the extravasation. When an aneurism is the cause of the hæmorrhage, certain symptoms, such as headache, vomiting, and double optic neuritis, may precede the onset. As a general rule, the phenomena attending the rupture resemble very closely, if indeed they are not precisely similar to, those which characterise the seizure in cases of extensive intra-cerebral hæmorrhage. The paralysis which accompanies the comatose condition nearly always affects all four extremities; it is rarely hemiplegic. Convulsions very frequently occur; but sometimes rigidity of the legs and arms is present. Vomiting is another somewhat characteristic symptom. Death often supervenes in a few hours.

In some cases the patients may not lose consciousness for some time after the hæmorrhage, as when the extravasation takes place slowly.

In the meningeal hæmorrhages of new-born infants the children are frequently born dead, or death occurs from asphyxia soon after delivery; but in some rare cases the infant remains lethargic or comatose for a few days, and then succumbs, generally in convulsions.

PROGNOSIS.

Death is the usual termination, whether in the case of adults or infants; but small hæmorrhages are not necessarily fatal.

TREATMENT.

In adults the general indications for treatment are the same in meningeal and intra-cerebral hæmorrhage; but in the case of infants efforts must be made to relieve the tendency to asphyxia, and with weak stimulants, friction, &c., the feeble heart must be excited to action.

CHAPTER VII.

CEREBRAL EMBOLISM AND THROMBOSIS.

WHEN the mass which occludes the vessel is carried to the site of occlusion from a distant part the process is termed embolism; when the obstructing clot is formed in the vessel it is termed thrombosis.

MORBID ANATOMY.

It may be accepted that the great majority of cases of so-called softening of the brain owe their origin to plugging of the cerebral vessels. The immediate result of the occlusion is the production of a bloodless or anæmic condition of the tract of brain substance supplied by the branches of the occluded trunk. The subsequent changes depend upon the vascular peculiarities of the part supplied by the affected vessel,—that is, upon the facilities which exist for the re-establishment of the circulation through collateral vessels. It will be recollected that the arteries which supply the basal ganglia, the “basal system,” are terminal vessels; while the vessels of the cortex freely anastomose with one another. Thus an embolus lodged in a

vessel, say the middle cerebral, which is supplied to parts which are not the seat of free anastomosis, will cause rapid and marked changes; but should the occluding mass find a resting-place in a vessel beyond the region of "terminal arteries"—in the cortex, for example—the collateral circulation may be sufficient to prevent the occurrence of serious pathological processes. When the seat of the obstruction is on the cardiac side of the circle of Willis marked brain changes seldom or never result, owing to the very free arterial inter-communication which exists.

The anæmic area soon becomes œdematous, and may be affected by marked hyperæmia with hemorrhages. These hæmorrhagic extravasations, mixed with the softened brain tissue, constitute "red softening." At times the œdematous tissue becomes softened without the accompaniment of hyperæmia and hæmorrhages, when, from its colour, it is termed "yellow softening."

Softening generally begins about the second or third day after the occlusion of the vessel.

After three or four weeks the reddish colour of the softened and hyperæmic mass begins to disappear, and the necrobiotic tissue becomes yellow, constituting another form of the so-called "yellow softening." The left middle cerebral artery is more frequently occluded by an embolus than any other cerebral vessel. It is doubtful if one artery

is more frequently affected than another by thrombosis, though it is said that the middle and posterior cerebrals and vertebrales are most frequently selected.

ETIOLOGY.

The fibrinous masses which constitute emboli are generally washed away from the aortic or mitral valves; sometimes they originate in the aorta itself, or in an aneurism of the first part of the arch. Occasionally the mass enters the left side of the heart from the pulmonary veins. In some rare cases embolism arises from the introduction of cancerous particles, caseous *débris*, or the solid contents of hydatid cysts into the circulation. Since endocarditis is the source of the fibrinous deposit on the valves of the heart, it is the most important predisposing cause of embolism. Diphtheritic endocarditis generally gives rise to multiple (capillary) embolism. The fibrinous masses are very often formed on a puckered valve apart from any recent inflammation.

Degeneration of the arterial walls stands in relation to arterial thrombosis as its commonest predisposing cause. As a result of the degenerating process (fatty degeneration, or atheroma) the lining membrane of the vessel becomes roughened and thickened, and its calibre lessened. Along with the alteration in the arterial wall, retardation of the

blood-flow plays a most important part in the production of thrombosis, and may obviously arise from fatty degeneration with dilatation of the heart, atheromatous thickening of the coronary arteries, with resultant impaired nutrition of the cardiac walls, and a number of general causes which tend to depress the circulation, such as the puerperal state, convalescence from acute diseases, &c. Syphilitic degeneration of the walls of the cerebral vessels seems especially prone to bring about coagulation within the artery. In many cases it is probable that the process commences in the very small arterioles, to the walls of which white blood corpuscles are apt to adhere under favourable conditions. A stoppage thus produced may spread to large trunks.

Thrombosis occurs more frequently in advanced age; embolism is met with oftenest between twenty and forty, although it may also occur in persons advanced in life.

SYMPTOMS.

Many of the features of an embolic attack are precisely the same as those of a cerebral hæmorrhage. The onset may be similar, and the resulting train of symptoms the same; but still in many cases a distinction can be drawn.

The symptoms of embolism can be discriminated in the early stages of the affection, but not in the later, the period of softening. When

the clot finds its way to a vessel from a distance (embolism), the symptoms which attend the occlusion are sudden in the extreme. In the case of thrombosis the onset is more gradual, and is often preceded by certain premonitory signs—*e.g.*, headache, giddiness, slight loss of power in an extremity with numbness, and other prodromata, —all of which may show themselves several weeks before the actual occlusion takes place.

In many cases of embolic plugging of the cerebral vessels there is no loss of consciousness; and in general, the comatose condition induced by embolism is more transient than that which attends a hæmorrhage. The exact nature of the symptoms, and distribution of the paralysis, which follow the seizure will of necessity depend upon the particular cerebral tract affected, and the size of the vessel occluded.

Aphasia is a very common, and in many cases the only, symptom of embolism, but more frequently right hemiplegia appears along with it.

A sudden attack of right-sided hemiplegia, ushered in by apoplectic coma of short duration, and accompanied by aphasia in a young person with a mitral or an aortic bruit, is characteristic of embolism of the left middle cerebral artery.

It is not uncommon to find the onset attended by epileptiform convulsions, which as a rule are general, but in some cases are limited to the para-

lysed side. These are very often met with in cases of bilateral embolism.

Thrombosis is generally preceded by premonitory signs, which arise from the gradual occlusion of the vessel and consequent diminution in the blood supply of the part to which the affected vessel is distributed. The final plugging of the vessel may be attended by loss of consciousness (apoplexy), or, as in the case of a good many embolic and some hæmorrhagic attacks, the onset may be entirely free from anything approaching to the comatose condition. It must be borne in mind, whilst due stress is laid upon the special diagnostic significance of premonitory symptoms, that cases of thrombosis occur at times without any previous warnings. The manner in which the symptoms of thrombosis develop very often suggests that not only is the affected area becoming gradually deprived of its blood, but also that the limits of the anæmic tract are widening. For example, the hemiplegia may commence in the leg, which becomes gradually more and more paralysed, the paralysis at length extending to the arm, until both arm and leg are markedly affected. The explanation of these events is to be found in the fact that the thrombus has a tendency to encroach upon an artery in a backward direction, and thus affects a wider area by plugging additional branches.

The stage of softening is generally marked at its

commencement by a rise of temperature. This elevation usually lasts a few days, and then a rapid fall takes place—a fall which is found to be much more rapid than usually marks the close of the period of inflammatory reaction in a case of hæmorrhage.

The subsequent features, supposing the anæmic tract to have become softened, resemble a case of hæmorrhage very closely. It is less common to meet with rigidity in embolism and thrombosis than in hæmorrhage; but it is now known that contractures occur more frequently than was formerly supposed in these affections.

Some authorities lay great stress upon the diagnostic aid furnished by an ophthalmoscopic examination. Not unfrequently, when the middle cerebral artery has become occluded, the retinal vessels are seen to be engorged, and the disc unusually red, and even blurred, owing to the ophthalmic artery taking part in the increased collateral flow. In some cases the cause of the cerebral attack may be inferred from the discovery that the ophthalmic artery has become occluded by a small embolus. In such a case the arteries of the retina are seen like fine threads, and, though of a red colour, yet appear to be empty. Evidences of splenic and renal embolism, which are undoubtedly very difficult to detect, are important as furnishing diagnostic help.

DIAGNOSIS.

Briefly, the following points indicate embolism :— early life, sudden attack without prodromata, implication of the left middle cerebral artery (right hemiplegia with aphasia), short duration or absence of coma, left-sided valvular disease, history of previous seizures of somewhat similar nature, and rapid recovery from the hemiplegia.

Thrombosis may be diagnosed from the occurrence of premonitory symptoms, slow development of the paralysis, advanced life, and evidence of diseased arteries.

The greatest difficulty in the direction of diagnosis lies in drawing a distinction, not between embolism and thrombosis, but between hæmorrhage and thrombosis.

PROGNOSIS.

The prognosis in occlusion of a cerebral vessel is always exceedingly grave. That such is the case will become especially apparent when it is considered that both embolism and thrombosis depend, in nearly every instance, upon most serious affections, the continuance of which makes it probable that similar attacks will recur. Any attempt to predict the result of a seizure at the onset will be found to be extremely hazardous.

TREATMENT.

Considering the risk we run in confounding the premonitory stage of thrombosis with that of hæmorrhage, it behoves us to be very careful what special prophylactic treatment we adopt, as stimulating measures, which theoretically would do good whilst a vessel is becoming occluded, would do immense harm in threatened hæmorrhage. It is better to urge a line of treatment suitable in every respect to cases of hæmorrhage, in which the following would form important items:—attention to diet, perfect quiet, and the judicious administration of mild laxatives.

When the diagnosis of embolism can be determined with certainty, the treatment should be of a stimulating and tonic character; but in all cases should be directed to the disease which underlies the attack. The treatment of the onset will be the same as in hæmorrhagic apoplexy; but attention may be called to the successful employment of the nitrite of amyl at this stage. Nitro-glycerine, also, from its similar physiological action, may be used.

CHAPTER VIII.

INFLAMMATION AND THROMBOSIS OF
THE CEREBRAL SINUSES.

THROMBOSIS of the sinuses arises either from inflammation of their walls (phlebitis), or from such alterations in the quality of the blood itself as, operating in conjunction with an enfeebled flow, are favourable to coagulation.

Inflammation of the sinuses is seldom primary, but results generally from disease of the cranial bones. Caries of the petrous portion of the temporal is especially prone to originate the inflammatory process; and, as suppuration of the middle ear is closely related to carious disease of the temporal bone, it follows that inflammations and purulent gatherings within the osseous portions of the ear are amongst the most frequent causes of inflammatory thrombosis of the cerebral sinuses.

Carbuncles and erysipelas of the face occasionally induce phlebitic coagulation in the sinuses (transverse and cavernous), the phlebitic process penetrating within the cranium by means of the facial and ophthalmic veins.

Thrombosis of the cerebral sinuses may, however, occur apart from local inflammations. In

some cachectic conditions of advanced life, attended by the alterations in the circulation above referred to, the process has been found to take place with tolerable frequency. In the *marasmus* of young children, also, it is an accident with which all who are engaged in the treatment of infantile disease are familiar. This form, depending upon general conditions, occurs most frequently in the longitudinal sinuses.

The immediate effect of occlusion of a sinus, from whatever cause, is stasis of blood in the affluent veins, which often contain thrombotic masses from extension backwards of the clot. Rupture of the veins may occur as a result of the stasis—an event which is occasionally marked by profuse meningeal hæmorrhage; however, in other cases the hæmorrhages are found to be small and capillary. Serous effusion is by no means a constant consequence of thrombosis, though it occasionally occurs both on the surface of the brain and in the ventricles. Meningitis very frequently attends thrombosis of the phlebitic variety; and in some cases the cortex suffers both in the direction of isolated patches of softening, and capillary hæmorrhages.

SYMPTOMS.

The diagnosis of thrombosis of the cerebral sin-
uses constitutes one of the most difficult problems

within the range of cerebral pathology. This difficulty arises in great measure from the complex nature of the lesion. The case may at the same time be marked by anæmia and hyperæmia of the brain; the former condition the result of the general affection on which the formation of the clot depends, as in the case of senile and infantile atrophic changes, and the latter due to the venous stasis. Again, meningitis, hæmorrhages, or localised softening, may be present; whilst the underlying affection itself must necessarily complicate the symptoms. Indeed, though there are a few symptoms of special diagnostic import, the majority of the features which characterise thrombosis of the sinuses are obscure, and belong in common to many cerebral affections.

As a rule the symptoms which result from occlusion of the superior longitudinal sinus (thrombosis of old age and infants) are more pronounced than those depending upon thrombosis of the sinuses of the petrous portion of the temporal bone (phlebitic occlusion),—a fact explicable on anatomical grounds.

In general terms, the symptoms of thrombosis of the phlebitic variety resemble the features of a case of septicæmia, with pronounced brain symptoms. During the course of an internal otitis, or in a case of severe inflammation of the upper part of the face, or after an injury to the head,

the following symptoms would point in the direction of thrombosis:—repeated rigours, with high temperature; considerable mental obtuseness, with perhaps quiet delirium; a marked typhoid condition, with dry tongue; and many others of a like character. Sometimes extreme headache is complained of, and motor paralysis and convulsions may be present.

The general features of a marasmic (cachectic) thrombosis vary according as the case occurs in the person of a young child, or an aged individual. Early in life the condition most frequently results from an attack of exhausting diarrhoea. Under such circumstances thrombosis, generally of the superior longitudinal sinus, is characterised, in addition to the already existing symptoms, in some cases by collapse and coma—resembling the hydrecephaloid of Marshall Hall, in others by rigidity of the muscles or paralysis, with perhaps convulsions. In this connection Nothnagel* writes:—"And we feel therefore justified in laying it down as a general rule that when diarrhoeas, occurring in children a few months old, are followed by cerebral disorders of the active motor kind just referred to [paralysis and convulsions], the diagnosis of thrombosis of the superior longitudinal sinus, so often involved under such circumstances, is more probable than that of simple cerebral anæmia."

* Ziemssen's "Cyclopædia," vol. xii., p. 215.

In cachectic thrombosis of adults the symptoms are extremely variable. In some cases apathy and general depression alone mark the onset. There is nearly always considerable mental defect, which may be of the nature of apathy, coma, or delirium. Motor disorders, though not constant, are frequent, and occur in the shape of tremor, convulsions, and paralysis. Sometimes the paralysis is most limited; the facial, or the third (motor oculi) may alone be affected.

The foregoing general features, both as regards children and adults, scarcely suffice for an accurate diagnosis; but when discovered along with the following characteristic ones, due to disturbances in the circulation, the case may readily be made out—viz., distension of the veins of the nasal passages, with resulting epistaxis; enlarged veins in the neighbourhood of the anterior fontanelle, temples, and ears; cyanosis of the parts of the face supplied by the anterior facial veins. The phenomena just mentioned may attend thrombosis of the superior longitudinal sinus.

When the lateral sinus is occluded, a puffy swelling (cedema) may be found, though rarely, to occupy the skin over the mastoid process.

In thrombosis of the cavernous sinus the eyeball occasionally stands out prominently, and the eyelid and conjunctiva may become cedematous.

PROGNOSIS.

Recovery takes place but rarely, the termination being almost always fatal. Occasionally cases of the kind appear to last several weeks, but death may occur within a few days.

TREATMENT.

Our treatment can only be prophylactic; that is, have for its object the removal of the conditions which are wont to give rise to thrombosis of the sinuses.

CHAPTER IX.

CAPILLARY EMBOLISM AND THROMBOSIS.

ALTHOUGH the symptomatology of occlusion of the cerebral capillaries is so vague and unsatisfactory as almost to make reference to the subject unnecessary in a practical clinical treatise, it will yet be expedient to allude briefly to the processes on which the occlusion depends.

Embolism of the capillaries may be the result of masses of pigment, fat or pus cells, perhaps white blood corpuscles, small particles from broken-up fibrinous clots, or bacteria, finding their way into the circulation.

Pigment-embolism has been found to occur, as a rule, in cases of ague. It is well known that in some chronic forms of intermittent fever degeneration of blood corpuscles takes place in the spleen, the result being that the cells are converted into blackish pigment-granules, which, when they occur in large quantities, constitute the condition known as melanæmia. It is in this condition of blood that pigment-embolism most commonly occurs.

Occlusion of the cerebral capillaries by oil globules may either occur from fat being formed

in the capillary itself, as the result of fatty degeneration of the wall of the vessel, or from the migration of the fat-drops from distant parts; the latter is undoubtedly the more usual mode of obstruction. These fat-emboli originate most frequently through the breaking-up of the atheromatous degeneration of the walls of the great arteries, the fatty contents being consequently swept into the circulation. In other cases they arise from absorption of the fatty matter of the marrow of the bones after fractures.

Thrombosis of the cerebral capillaries occasionally arises from calcareous deposit—"lime-metastasis" of Virchow—in the walls and in the interior of the vessels. Virchow suggests that the mineral matter is absorbed from the bones, and then, owing to a failure on the part of the kidneys to excrete it from the blood, it becomes deposited in the brain.

The nature of the changes in the brain as the result of capillary occlusion depends in great measure upon the number of vessels affected. If these be few the vascular disturbance may be extremely slight; if numerous, multiple cerebral softening may result.

It is probable that minute abscesses occur from the lodgment of specific (septic) emboli in the capillaries.

As already remarked, the symptoms of capillary

occlusion are by no means characteristic; but such an occurrence may be reasonably suspected if pain in the head, vertigo, delirium, convulsions, or paralysis make their appearance during or subsequent to a malarial fever. In the case of fat-embolism, the symptoms are generally much less pronounced than in fat-thrombosis; in the latter condition the affected tract is wont to be more extensive than in the former.

Mental disturbances are often well marked in capillary occlusion, but on the whole the symptoms are general and indefinite; giddiness, nausea, headache, with more or less paresis, are among the commonest.

The prognosis depends altogether upon the extent of the affected area.

The remedial measures must be employed entirely on general lines.

CHAPTER X.

CEREBRAL HYPERÆMIA.

WE are unable at the present time to accept the conclusions of Munroe and Abercrombie, who maintained that the adult skull inclosed a constant quantity of blood. More recent inquiries have revealed the fact that the cerebral circulation is variable.

Two forms of congestion of the brain are met with:—the *active*, “active arterial hyperæmia”; and the *passive*, “passive venous stasis.”

MORBID ANATOMY.

Increased tension within the cranium is the first and probably the immediate effect of hyperæmia, whether active or passive.

In intense congestion the vessels of the pia mater are distended with blood, and the dura mater may be of a bluish tint. The brain is generally enlarged; and on section the white substance presents a large increase of the vascular puncta, both as regards size and number; in very marked cases it may be of a reddish colour. The gray matter is generally darker than usual, and small hæmorrhages may be seen in it.

In some cases the congestions are local. The cortical substance may present hyperæmic appearances, whilst the white matter remains anæmic. Some of the central ganglia may alone be affected.

In chronic cases the meninges often become opaque and thickened, and the cortical matter discoloured from pigmentation.

A venous stasis is often, as Nothnagel has shown, attended by an arterial anæmia; indeed, in general terms it may be assumed that a venous hyperæmia means a lessened arterial flow. In very chronic cases the ventricular fluid is increased and the pia mater cedematous.

ETIOLOGY.

All conditions which raise arterial tension tend to produce active hyperæmia of the brain; such as increased activity of the heart, application of cold to the surface of the body, and certain medicines. However, it rarely happens that hyperæmia, occurring as the result of such causes, produces symptoms.

Certain gastric disturbances are accompanied by active cerebral congestion, due to vaso-motor irritation (reflex). Mayer and Pribram have shown that electrical or mechanical irritation of the walls of the stomach produces a reflex increase in the arterial tension. Along with this it is probable that the irritation from the stomach has the

effect of actively dilating the vessels in the region of distribution of the carotid through the vasodilator nerves. Well-marked symptoms of cerebral hyperæmia occur in some cases after a full meal, without any increase in the rate or strength of the cardiac beats. Blows on the head undoubtedly induce active congestion, and many of the symptoms resulting from such an injury are due to this cause.

According to Andral and Niemeyer, prolonged and intense mental activity is a cause of congestion of the brain; others deny that such is the case, and, indeed, it seems probable that the symptoms which arise from prolonged mental strain are due rather to the unusual functional activity than to hyperæmia.

The most prominent causes of *passive venous stasis* are to be found in organic diseases of the heart. Tricuspid regurgitation, whether primary or dependent upon mitral disease, produces very marked passive congestion of the brain. Pulmonary disease, when attended by much obstruction to the flow of blood through the lungs, must necessarily have the same effect. And in like manner, aneurisms, or tumours within the thorax, interfering with venous flow from the head and neck, often give rise to pronounced cerebral symptoms.

SYMPTOMS.

As a rule the *active* form is attended by more or less excitement, whilst depression may be said to characterise the *passive* variety.

In moderate cases giddiness, noises in the ears, with a peculiar sense of confusion and headache, are often complained of. The patient is unable to sleep without disturbing dreams, or he may be unable to sleep at all during the attack. His manner is generally changed in the direction of irritability. There may be nausea or vomiting.

In the more severe attacks these symptoms are exaggerated; the pulse becomes hard, full, and persistent, the carotids pulsate, and the pupils are generally contracted. An inability to settle for even a few minutes is often observed, and the patient is restless and erratic. Great excitement frequently prevails, amounting, it may be, to active delirium. The face is livid, and the symptoms are practically those of meningitis and cerebritis. Convulsions in adults are not common, but in the case of children they occur frequently.

The worst attacks occur in the form of apoplectic seizures,—the so-called “apoplexia e plethora,” or “coup de sang.” In such cases consciousness is generally regained in a few hours, and the patient recovers without any paralysis. In some cases, however, weakness of one or more limbs has been found to follow the attack.

TREATMENT.

In all cases the cause of the hyperæmia must be combated. Complete quiet and, as a rule, confinement to bed are necessary.

In active congestion aconite may be useful. Cold should be applied to the head, and saline purgatives administered. Mustard pediluvia, or flannels wrung out in hot water and mustard and applied to the lower extremities, often do good. The diet should be restricted and exceedingly plain.

Venous congestions are often benefited by bleeding; the abstraction of a moderate quantity generally suffices. Active purgatives, suitable diet, and rest are the most reliable remedial measures.

CHAPTER XI.

CEREBRAL ANÆMIA.

MORBID ANATOMY.

It often happens that whilst the brain substance is pallid in cerebral anæmia, the meningeal vessels, and especially the large venous trunks and the sinuses of the dura mater, are well filled, as was the case in a woman who died recently from divided right internal jugular vein, on whom the writer made a post-mortem examination. In some cases the membranes are also exsanguineous.

The cortical gray matter is often so pale as to be with difficulty distinguished from the adjacent white substance. The latter is more or less dry and firm, though it may be soft in consistency and moist, *e.g.*, when the anæmia is dependent upon or accompanied by cedema. The anæmia is prone to be partial when it is connected with localised lesions of the brain, as tumours, embolism, thrombosis, and hæmorrhages.

In certain cases of chronic anæmia the result of some general disorders, especially cirrhotic kidney, the pia mater is markedly cedematous, and the ventricles are moderately filled with fluid.

ETIOLOGY.

Severe hæmorrhage is a very common cause of anæmia of the brain: Chronic and exhausting discharges, and other states productive of general anæmia, such as chronic dysentery, albuminuria, empyema, leucocythæmia, progressive pernicious anæmia, cancer, &c., also induce a cerebral anæmia. In the former case, should the loss of blood be sudden and copious, the anæmia will occur as an acute affection; but on the other hand, where there is a gradual drain on the system, a chronic anæmia will be the result.

Lesions of the aortic valves, especially insufficiency, are very frequently responsible for cerebral anæmia; and feebleness of the heart through fatty degeneration may also act in a similar manner.

Embolism and thrombosis are common causes of the condition, occasioning as they do, in nearly every case, a partial anæmia which may be either temporary or permanent. Temporary diminution in the calibre of the cerebral arteries, either alone or accompanied by temporary cardiac inhibition, underlies the cerebral anæmia on which ordinary fainting attacks depend.

SYMPTOMS.

Syncope is the most prominent symptom of acute general cerebral anæmia. In exaggerated cases, as when a very large quantity of blood is rapidly lost,

convulsions, epileptiform in character, make their appearance. In some chronic cases convulsions may occasionally be met with.

But besides fainting and convulsive fits, cerebral anæmia gives rise to many other symptoms almost as distinctive, though much less alarming. Headache is often present; indeed, anæmia of the brain is one of the commonest causes of headache. The pain is most frequently frontal, though it often affects the occipital region. Vertigo and ringing in the ears are complained of; the former is markedly increased on rising from the recumbent position.

Vomiting, dimness of vision, drowsiness, subjective sensory phenomena—"needles and pins," &c., reeling gait, enfeebled mental power, anomalous neuralgic-like pains, are all met with from time to time in cases of chronic cerebral anæmia.

In infants wasting diseases, especially severe attacks of diarrhoea, will often induce a markedly anæmic condition of brain, the *hydrocephaloid* or *hydreencephaloid* of Marshall Hall. After an initial stage of irritation characterised by restlessness and fretting, the symptoms of this affection, in which the anæmia is accompanied by effusion into the ventricles, are as follows:—the child seems inclined to sleep almost constantly, the features are pinched and pale, the pupils, though at first small, become large and almost immovable, the pulse is small and weak, and the fontanelle, if still open, is depressed; should

the case advance unfavourably, coma will supervene, and death be the result.

DIAGNOSIS.

As it is now known that the optic papillæ may become swollen in intense anæmia, and as cerebral anæmia is often attended by headache, vomiting, and reeling gait, it may be a matter of difficulty to distinguish the condition we are considering from intra-cranial tumour. Cerebral anæmia may be readily mistaken for hyperæmia, but a history of the case and a careful examination into the general condition of the patient will do much to aid in drawing a distinction. In this connection the ophthalmoscope will prove useful, for in the anæmic state the retina will be pale and the reverse in the hyperæmic. In anæmia the headache is generally localised; in congestion it is diffused. The drowsiness of the anæmia, set off against the insomnia of the hyperæmic condition, will also aid.

The prognosis depends entirely upon the nature of the condition which underlies the anæmia.

TREATMENT.

Removal of the cause of the anæmia, such as the arrest of hæmorrhages or other exhausting discharges, is necessarily the first indication to be fulfilled. The horizontal position should as far as possible be insisted upon. Stimulants, especially

in alcoholic form, are generally called for. The diet should be liberal and stimulating, unless contra-indicated by acting injuriously upon the underlying affection, as in the case of ulcer of the stomach. When much cerebral excitement (delirium) is present opium should be given, though with caution. Transfusion may in some cases of anæmia from loss of blood offer the only chance of recovery. Nitrite of amyl is often used in relieving attacks depending upon cerebral anæmia, and is especially useful in anæmic headaches. Iron, quinine, digitalis, and ammonia are the pharmacopœial remedies most frequently employed.

CHAPTER XII.

DISEASES OF THE DURA MATER.

INFLAMMATION of the dura mater of the brain, or pachymeningitis cerebri, has been divided by Virchow into two forms, the external, and the internal.

EXTERNAL PACHYMENINGITIS.

In this affection the inflammation affects the external layer of the dura mater.

MORBID ANATOMY.

Early in the process there is congestion on the outer surface, generally accompanied with ecchymoses, and followed by the usual inflammatory exudation, which results in thickening and adhesions. It is not unusual in chronic cases to meet with osseous lamellæ in the thickened portions of the dura mater and in the new-formed adhesions. In some cases suppuration occurs, and then the purulent matter generally finds its way to the internal surface of the membrane; but at times the pus accumulates between the bone and the dura mater, disintegrating the latter and separating it from the cranium. The process may extend to the sinuses, causing thrombosis.

ETIOLOGY.

External pachymeningitis is most frequently met with after *injuries* to, and *caries* of, the cranial bones. Disease of the petrous portion of the temporal bone is very often the starting-point. In other cases the process extends from the cribriform plate of the ethmoid, or it may originate in caries of the first cervical vertebra.

SYMPTOMS.

The symptoms are often obscure, but such as are present should always be viewed along with the etiological considerations—wounds, caries of the bones of the skull and upper cervical vertebræ, &c. Headache, often circumscribed, is an important symptom. Vomiting, giddiness, and convulsions may be present; but as a rule, by the time the case presents severe symptoms the process has extended beyond the external lamella of the dura mater, and the affection can no longer be regarded as simple external pachymeningitis.

TREATMENT.

Should the commencement of external pachymeningitis be suspected, absolute rest with restricted diet must be enforced. Cold applied to the head and leeches to the mastoid process

prove serviceable; and at the same time purgatives should be administered.

INTERNAL PACHYMENINGITIS

(Hæmatoma of the Dura Mater).

Two varieties of inflammatory processes have been described as attacking the internal lamella of the dura mater, the purulent and the hæmorrhagic. The former, being closely allied to the purulent inflammations of the adjacent coverings of the brain, calls for no special description; the latter deserves attention, however, as it can be recognised clinically as a distinct affection.

MORBID ANATOMY.

The first morbid change is characterised by congestion, as the result of which an exudation takes place on the surface of the dura mater. The cells of the exudation become early changed into connective-tissue corpuscles, and a false membrane is thus formed, composed of loose connective tissue. Into this new membrane a large number of capillaries grow, many of which present varicosities. These vessels are prone to rupture, and blood escaping into the spaces of the loose spongy tissue constitutes the so-called *hæmatoma*.

This hæmorrhagic deposit occurs very frequently

on the under surface of the dura mater beneath the parietal bones, and is sometimes large enough to compress and flatten the convolutions.

According to Huguenin, the extravasation of the blood takes place directly from the surface of the dura mater without the formation of an exudation membrane.*

ETIOLOGY.

Injury and chronic alcoholism are frequently responsible for hæmatoma of the dura mater, and old age is an important predisposing cause. It sometimes occurs in acute rheumatism, typhus and typhoid fever, acute tuberculosis, and particularly in general paralysis of the insane.

SYMPTOMS.

Most of the cases run a chronic course. The symptoms, especially when the affection occurs in old people, are very suggestive of cerebral softening. Perhaps the most constant symptom is headache, which is persistent, and though often general, is at times confined to the region of the hæmatoma. The patient will generally complain sorely when the affected area is percussed, and manifest a weak intellect and a tendency to drowsiness. Hemiplegia may be present, but sometimes all four extremities are more or less

* Ziemssen's "Cyclopædia," vol. xii., p. 386 (English trans.).

affected. Occasionally muscular rigidity and twitchings are observed; and there may be inco-ordination and unsteady gait. The pupils are generally contracted, but dilatation will occur in consequence of compression from a large hæmorrhage. Sudden relapses mark the occurrence of fresh hæmorrhages.

PROGNOSIS.

The prognosis is undoubtedly very unfavourable; but at the same time it should be recollected that the remnants of old-standing hæmatomata are occasionally found on the surface of the brains of patients who have died from entirely different affections.

TREATMENT.

There is little to be done beyond keeping the patient perfectly quiet. Cold to the head may be required in the early stages, or when additional hæmorrhages are suspected. Counter-irritation has been recommended in the later periods of the disease. The treatment will, however, depend in a great measure upon the cause of the internal pachymeningitis.

TUMOURS, OR NEW GROWTHS occasionally have their starting-point in the dura mater. They may be cancerous (particularly the endothelioma variety), sarcomatous (generally small round-celled sarcoma), syphilitic, osseous (osteomata), or present the characters of the psammoma. The clinical recognition of tumours developed in this membrane is exceedingly difficult apart from the general diagnosis of intra-cranial tumour, unless the growth should perforate the skull, which not unfrequently happens in connection with sarcoma.*

* See a paper on the Diagnosis and Nature of Perforating Tumours of the Dura Mater by the writer: *Brit. Med. Jour.*, Oct. 13th, 1883.

CHAPTER XIII.

TUBERCULAR MENINGITIS

(Miliary Tubercle of the Pia Mater).

MORBID ANATOMY.

THE small tubercular granulations, though occasionally found in the pia mater covering the convex surface of the brain, attack, in the great majority of cases, the basal portion of the membrane, selecting most frequently the fissure of Sylvius; by a careful separation of whose lips the deposit in typical form may generally be discovered.

The granulations are met with in the course of the vessels of the pia mater. The membrane is more or less inflamed, and the inflammation is generally accompanied by exudation of a serous, plastic, or purulent character. The choroid plexuses are commonly affected, though the number of gray granulations to be found in them is seldom very large. The lateral ventricles are generally distended with serosity, which frequently permeates and dilates the aqueduct of Sylvius, and invades the fourth ventricle. The deposition of miliary tubercle in the pia mater is often associated with a similar deposit in the lungs, peritoneum, liver, and other organs.

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ETIOLOGY.

A scrofulous diathesis is an important predisposing cause. Unfavourable hygienic conditions can scarcely be over-estimated as tending to further the development of the disease. In the first decade of life most of the cases occur, and the disease may be said to be especially frequent between two and seven years; it is very seldom met with after forty-five, between twenty and forty being the most susceptible period for adults.

SYMPTOMS.

A great variety is observed in the clinical manifestations of this disease. The affection may commence insidiously, or the patient may, in the midst of apparent perfect health, be suddenly attacked by symptoms suggestive of acute inflammation of the basal pia mater.

The cases characterised by a premonitory stage are most frequently found in children, whilst in adults the sudden onset is more common. Yet it must be recollected that a large number of cases of tubercular meningitis are secondary to pulmonary phthisis, mesenteric disease, chronic peritonitis, scrofulous disease of the glands, bones, &c., and to a number of other affections attended by more or less constitutional disturbance, the presence of which would necessarily mask the prodromata.

The prodromous stage, as in a child, is commonly characterised by a complete alteration of the patient's manner, peevishness and gloominess, and disinclination to his usual amusements. He is restlessly disposed, though sluggish in his movements. The appetite is lost, and vomiting may occasionally occur. The child is drowsy, but sleeps badly, and often with its eyes partially open. The evacuations become disordered. Constipation is the rule, though alternated at times with attacks of diarrhoea. The tongue is usually white in the centre, and red at the tip. Progressive emaciation, from which the face is often exempt, is nearly always observed.

Headache and giddiness are generally present, the latter evidenced by the child "suddenly standing still, gazing round for a moment as if lost, then either beginning to cry at the strange sensation or seeming to awake from its reverie, and at once returning to its play" (West).

The premonitory stage of the affection in adults is marked by a general feeling of malaise, headache with the sensation of cephalic constriction and giddiness, sleeplessness, impaired nutrition, feverish sensations with increased pulse and temperature, and so forth.

This stage sometimes lasts for several weeks before the appearance of the urgent inflammatory symptoms. As the affection further advances the

symptoms above mentioned become more prominent, until the occurrence of the following clinical phenomena, significant of the full development of the lesion so far as miliary tubercles and inflammation are concerned, are observed as the most constant symptoms of the onset, viz., headache, vomiting, intolerance of light, and, in the case of infants, convulsions.

The headache is seldom confined to any particular region, though the front of the head is generally indicated as being the seat of the most intense pain. It is often paroxysmal, as shown by the patient's sudden cries and moans. Giddiness is seldom absent; while in some cases the sensation of whirling round, or of falling, is almost perpetually experienced.

The pulse and the temperature are very variable. At first the former may be slow and full, becoming generally more rapid, and, it may be, irregular as the case advances; the charts of the latter are by no means uniform, and present nothing typical.

The respirations are frequently extremely irregular. Convulsions of a partial character, muscular rigidity, grinding of the teeth, retraction of the abdominal muscles, conjugate deviation of the eyes or convergent squint, appear from time to time. In children general convulsions may recur daily from the period of invasion, but in the case of adults the convulsions are seldom epileptic in character.

Too much stress should not be laid upon the “*tache cérébral*,” *i.e.*, red line left by the finger when drawn along the skin, for although it is a sign of importance (Trousseau), it cannot at the same time be regarded as pathognomic of tubercular meningitis, being occasionally present in other affections.

The “hydrocephalic cry,” generally supposed to be independent of consciousness, has also been observed in other acute diseases; it is a single loud cry, half-whine and half-shriek.

The eyes should always be examined with the ophthalmoscope; occasionally tubercles may be discovered in the choroid, though their eruption here is rare except in cases of general military tuberculosis. Optic neuritis is seldom met with. According to Cohnheim and others, “certain varicosities of the retinal veins, retinal hæmorrhages, and a serous peri-papillary infiltration (signs of obstructed circulation at the chiasm*)” are to be seen in some cases.

As effusion takes place the signs of cerebral compression appear; the patient becomes more and more unconscious until complete insensibility is observed. Cutaneous sensibility and the superficial reflexes which were previously exalted are extinguished. The paralysis becomes general, the

* Rosenthal, “Diseases of the Nervous System” (English trans.) vol. i., p. 23.

flaccidity of the limbs is only varied by the occasional occurrence of a convulsion, especially frequent at this stage in children. The pupils are dilated and fixed. Often the muscles of the back ~~of the neck and jaws~~ are rigid. In the case of young children the ~~fontanelle is full~~ and throbbing; but as death approaches the pulsations ~~generally~~ cease almost entirely.

DIAGNOSIS, PROGNOSIS, AND DURATION.

Tubercular meningitis may generally be distinguished from tumour of the brain by the absence of well-marked double optic neuritis, the acuteness of the invasion, and the history of certain affections already mentioned which are apt to forerun the tubercular disease.

From cerebro-spinal meningitis the tubercular form is to be diagnosticated by the absence in the case of the latter of a *severe initial chill*, pain in the nape of the neck and back, &c.

Though the diagnosis between typhoid fever and tubercular meningitis is often difficult, especially in children, the characteristic temperature chart, the painful and tympanitic abdomen, the looseness of the bowels, and the enlarged spleen of the former serve to distinguish them.

“Hydrocephaloid” disease (*see* p. 112) has often been confounded with it from forgetfulness of the fact that the former is generally characterised by

extreme depression and quiet somnolence, along with depressed fontanelle.

From simple meningitis, either basilar or of the convexity (purulent), the tubercular variety may be thus distinguished:—the onset of **the former affections** is generally **sudden**, and without any prodromous period; simple meningitis is accompanied by greater febrile disturbance, and by earlier and more acute delirium than tubercular.

Tubercular meningitis often simulates many other affections than those mentioned above; and, indeed, cases from time to time arise in which a correct diagnosis cannot with any degree of certainty be arrived at. For example, hysteria, delirium tremens, leptomeningitis infantum, abscess and hyperæmia of the brain, and thrombosis of the cerebral sinuses, are often confounded with it.

Trousseau lays great stress upon irregularity of the respirations as pointing to tubercular meningitis.

The prognosis is exceedingly unfavourable; the disease if not invariably fatal, is almost so, and indeed the few cases that *are claimed* as recoveries form the rare exception to the rule.

The average duration, excepting the premonitory period, in uncomplicated cases is from ten to fifteen days; occasionally the disease lasts several weeks, but death may occur in the first week.

TREATMENT.

Treatment can be of little avail in a disease distinguished by so discouraging a prognosis. In all probability prophylactic measures alone are of service, and these are necessarily only directed against tubercular meningitis in common with many other cognate diseases prone to arise in a scrofulous diathesis. After the development of the meningeal inflammation we have little to rely upon; leeches may be applied to the temples, and cold to the shaven scalp. In some cases warm applications appear to soothe, especially after effusion has taken place. Blisters are, in my opinion, deleterious.

Niemeyer recommends iodide of potassium, but most writers condemn it as useless. Chloral will generally relieve the convulsions, and may be injected into the rectum; five or seven grains so administered will prove sufficient in the case of a child of two years to arrest the fits for several hours.

CHAPTER XIV.

SIMPLE MENINGITIS.

BASILAR MENINGITIS.

PRIMARY inflammation of the pia mater at the base of the brain—*i.e.*, not tubercular, and independent of other affections likely to cause such a lesion, such as tumour, abscess, phlebitis of the sinuses, bone disease, &c.—is an affection of early adult life, and one that occurs but rarely. According to Hammond syphilis and alcohol are its commonest causes when chronic.

The intensity of the inflammatory process varies; some cases are acute, others are protracted, but in general the disease pursues a chronic course.

MORBID ANATOMY.

In the acute cases the pia mater at the base is the seat of fibrino-purulent deposit. In some cases the exudation coats several of the cranial nerves matting them with shreds of lymph to the adjacent thickened pia mater, and involves the choroid plexuses. There is often a considerable quantity of ventricular effusion. In more chronic

cases the membranes are cloudy and thickened, and the neighbouring cerebral tissue is softened and œdematous.

SYMPTOMS.

Headache, giddiness, and vomiting or nausea are generally complained of early in the course of the attack. The pain may be felt in the face and mistaken for ordinary neuralgia. The vertigo may be so intense as to interfere completely with locomotion. In some cases, though rare, no paralysis exists. The motor disturbances are, as a rule, irregular, and embrace the occasional but rare affection of the facial or of the hypoglossal nerve, more commonly that of the abducens, or of the third completely or in part, as well as an impairment of deglutition, and occasionally a paralysis of one or more of the extremities, which is seldom complete.

One paresis disappears and gives place to another; contractures and spasms of the nature of trismus, retracted abdomen, and stiffness of the neck occur, but are generally short-lived, so that it is not at all unusual to find the clinical picture changing from time to time.

Hyperæsthesia of the face or of different parts of the body is common enough. Anæsthesia is comparatively rare.

The mental faculties are often impaired. Optic neuritis has been observed with tolerable frequency.

The temperature is irregular; early in the attack it is generally high, especially at night; later on it sinks and remains low. The pulse generally agrees with the temperature in indicating a condition of high fever during the initial period, and depression towards the termination.

PROGNOSIS AND DURATION.

The prognosis is very unfavourable, though not absolutely hopeless. Some cases linger on for two or three months, or even much longer, and others run a rapid course. One case referred to by Huguenin ended fatally in seventeen days.

TREATMENT.

Iodide of potassium is often employed, but it is doubtful whether its administration is ever attended with any benefit except in syphilitic cases. The most efficient remedies are perhaps local blood-letting and cold applications early in the course of the disease, and blisters later on. Chloral and morphia may be used to relieve headache, if intense.

MENINGITIS OF THE CONVEXITY.

Though the distinction between meningitis of the base and that of the convexity cannot in

every case be drawn with rigid anatomical accuracy, yet it is one of practical clinical importance.

MORBID ANATOMY.

The pia mater becomes adherent to the cortex, which is commonly more or less softened and friable. The exudation is often discovered to be purulent; the pus, which may extend over both hemispheres, and not unfrequently encroaches on the base, generally selects the tracts of the vessels. In some cases ventricular effusion is present in considerable quantity. In general paralysis of the insane, in epilepsy, syphilis, and other affections more or less accompanied by chronic meningitis, along with the adhesions to the surface of the brain thickening and fibrinous exudations are usually met with.

ETIOLOGY.

Primary meningitis of the convexity is undoubtedly rare. It may occur at all ages; but according to Bierbaum, it chiefly attacks infants under two years. It then diminishes up to youth, when it again increases in frequency; but, excepting the chronic form, is less often observed in advanced life (Rosenthal). Its causes are very imperfectly understood.

The secondary variety often occurs as an ex-

tension of the inflammatory process from the dura mater, and may appear as the result of external injury, caries of the cranial bones, especially the temporal, internal otitis, syphilis, erysipelas of the face and scalp, carbuncles of the face, and old-standing intra-cerebral disease, such as tumours, abscess, &c.

SYMPTOMS.

From the numerous and diversified causes of meningitis of the convexity it is easy to understand why the cases present such widely different symptoms, both as regards the onset and the course. Some cases begin acutely and run a rapid course; others are insidious in their origin, and comparatively slow in their development and progress. The former type unquestionably prevails. Should the case not begin suddenly, but be preceded by premonitory features, headache, at times violent, general feverish sensations, sleeplessness, giddiness, apathy, and other symptoms more or less difficult to elicit are complained of. In the case of secondary meningitis the premonitory signs may be masked entirely by the symptoms of the underlying affection. Most frequently the meningitis manifests itself from the beginning in a sudden and pronounced way, the brain symptoms being quickly developed and unmistakable. Intense headache, giddiness, vomiting, and photo-

phobia, preceded, it may be, by a rigour, or in the case of children by a convulsion, mark the ordinary commencement. Sometimes, though rarely, with but scant warning the patient is seized with most excruciating pain in the head, and almost at once passes into a state of somnolence or wild delirium.

These initial symptoms are frequently followed by a peculiar mental condition characterised by irritability, restlessness, somnolence, delirium more or less marked, and tendency to apathy. Cutaneous hyperæsthesia, with well-marked superficial reflexes, is also noticeable. An indication of fever is given by an increase in the temperature, pulse, and respirations which are often irregular. The earliest motor disturbance, unless the case be ushered in by convulsions, occurs in the form of reeling gait, which is due most probably to the sensation of giddiness. Later on there may be convulsions either partial or general, followed by loss of power (muscular paralysis). Retraction of the head, in some cases amounting to opisthotonos, through rigidity of the muscles of the back of the neck, trismus and grinding of the teeth, strabismus, retraction of the abdominal walls, and many other evidences of local spasm make their appearance.

Facial paralysis occurs with tolerable frequency in cases due to purulent otitis, the paralysis being

on the same side as the ear affection. Sometimes the hypoglossal of the same side is affected. The fifth and the various nerves supplying the eye-ball may also be engaged should the inflammation (as is frequently the case) extend forwards at the base.

Beyond fulness of the veins, and occasionally some swelling of the papillæ, nothing very distinctive can be made out with the ophthalmoscope.

The pulse, at first rapid and full, becomes slow when effusion takes place, but very rapid in the last stages of the malady.

Towards the last the somnolent and paralytic symptoms predominate, and profound coma from which the patient cannot be roused forms the closing scene of the case.

PROGNOSIS.

Simple, non-tubercular, purulent meningitis is very often fatal; and though a few cases appear to recover, especially when the inflammation is associated with an abscess the contents of which escape externally, a very grave prognosis should be made.

Some cases terminate in a day or two from the commencement of the symptoms, but in others the patient may linger on for two or three weeks.

TREATMENT.

Leeches, with cold applications to the head, are generally employed early in the course of the affection. Purgatives are commonly required, though irritating remedies of this nature should be avoided. Iodide of potassium has sometimes been used with benefit, and Hammond suggests that it should be combined with bromide of calcium. I confess I have lost faith in the bromides in meningitis, and believe chloral is much more to be relied upon, and may be used to allay restlessness. Morphia is at times required when the headache is very severe.

CHAPTER XV.

HYDROCEPHALUS.

ALTHOUGH only a symptom of disease, in the sense that dropsies elsewhere are symptomatic of some prior disorder, we are justified in regarding hydrocephalus as a special affection, just as it is expedient to look upon pleural effusion as a disease demanding particular notice.

Hydrocephalus is either acute or chronic. Acute hydrocephalus has long passed as a synonym for tubercular meningitis, but the latter affection, having been already considered, need only be referred to here as a cause of the former.

LEPTOMENINGITIS INFANTUM

(*Acute Hydrocephalus*).

Leptomeningitis Infantum is the name given to the pathological processes that underlie acute hydrocephalus when occurring in young children, and *not tubercular in origin*. It is by no means clear that this form of acute hydrocephalic effusion is always the result of inflammation; at least it has been conceded by nearly all writers that cases arise, and not unfrequently, which present clinical phenomena justifying the diagnosis of acute

intra-ventricular effusion, and verified after death without any distinct appearance of pia-matral inflammation.

It occurs most frequently in children under five years. The effusion may result from the cerebral hyperæmia which often attends the processes of dentition; or arise in the course of scarlatina, measles, and the other eruptive fevers. Blows on the head have been known to occasion it; and, lastly, the ingestion of alcohol (Goll), and severe diarrhœa are recognised as exciting causes of the disease.

MORBID ANATOMY.

The effusion is always within the ventricles, which are often greatly distended. The pia mater generally appears normal to the naked eye; rarely has it been found even congested. The fontanelle is prominent, and the cranial bones may be considerably separated. There is often a marked flattening of the convolutions with more or less obliteration of the sulci. The choroid plexuses are usually hyperæmic and enlarged, and are the seat of minute hæmorrhages.

SYMPTOMS.

The clinical history resembles that of tubercular meningitis very closely.

In *leptomeningitis infantum* there is seldom a distinct prodromous stage, and when it does exist

it is usually short. The febrile condition, often observed from the commencement, may be entirely overlooked owing to the presence of a like state previously established, and dependent upon dentition, scarlatina, &c. A convulsion is frequently the first indication in young children of the new disorder. Vomiting occurs, and constipation is rarely absent. The child is uneasy and fretful. The eyelids are kept more or less shut in order to exclude the rays of light from the hyperæsthetic retina. During sleep they are not entirely closed, and the eyeballs are seen to roll from side to side beneath the upper lids; the pupils are contracted but movable. The fontanelle, although not distended, is observed to pulsate strongly. The skin is hyperæsthetic. Frequent twitchings of the arms and legs are present. Children able to describe their symptoms complain of headache, and younger patients make it apparent that they are suffering from the same by moaning, knitting the brow, and rolling the head about. These symptoms generally last for two or three days, when they give place to others of a more pronounced character. The child, who was previously affected by sleeplessness, passes into a condition of drowsiness or stupor. Or an attack of general convulsions ushers in a state of complete insensibility. The pupils are now dilated and fixed; the fontanelle distended, and the pulsations less marked. The hydrocephalic cry is often heard

at this stage. The convulsions may recur at short intervals, or continue with but slight intermission until the end. The respirations and pulse show a marked irregularity; and the child may die in a convulsion, or sink into a condition of profound coma from which it is never aroused.

PROGNOSIS, DURATION, &C.

The majority of the cases terminate fatally within a fortnight. Some few, however, appear to recover; but recovery is very rarely complete, generally being attended by contractures and impaired mental condition. One case I had the opportunity of making a post mortem upon a year after the commencement of the attack, which had evidently passed into a condition of chronic hydrocephalus, showed chronic ventricular effusion as the only morbid appearance.

The DIAGNOSIS from tubercular meningitis is, as all must allow, very difficult. In *leptomeningitis infantum* the prodromal stage is generally wanting, whilst tubercular hydrocephalus in children is nearly always characterised by an initial period of warning. In the former affection the convulsions seem to be more persistent, and to occur earlier than in the latter, in which their advent is usually at a later stage. Should the patient recover, the case is more likely one of simple than of tubercular hydrocephalus.

The TREATMENT should be conducted on the same lines as already laid down in the case of tubercular meningitis.

CHRONIC HYDROCEPHALUS.

Two varieties of this condition have been described, the internal and external, or a serous accumulation which occupies either the ventricular cavity, or the so-called sac of the arachnoid. The latter, on account of its extreme rarity, may be entirely disregarded; the following remarks will refer wholly to ventricular effusion.

MORBID ANATOMY.

The cranial bones are frequently thin and membranous, and the sutures widened; but in some cases the skull is actually denser and thicker than normal. The convolutions are flattened, and the sulci effaced, and in exaggerated cases the brain substance is thinned out, and the white and gray substance with difficulty distinguished. The effusion, which varies in quantity from three or four ounces to several pints, will, when copious, on the removal of the skull-cap, often lacerate the attenuated brain substance and escape. The cerebral substance that forms the walls of the dilated ventricles, though sometimes tough and resisting,

is generally softened, and the fornix, septum lucidum, &c., are sometimes broken into shreds. If the septum be unbroken, the foramen of Monro between the two cavities is found dilated.

The spinal canal not unfrequently shares in the process, the two cavities communicating freely.

In congenital hydrocephalus abnormal openings are sometimes formed in the skull, through which the brain may project under the skin, constituting *hydrancephalocele*.

ETIOLOGY.

Chronic hydrocephalus may be either congenital or acquired. If the latter, it may be either of inflammatory or purely mechanical origin. In old age it may be compensatory to atrophic changes. The causes of congenital ventricular effusion are very imperfectly understood. Rickets and syphilis are asserted to be the most frequent predisposing causes. A large number of the cases met with amongst children are congenital, although it is often difficult to elicit a conclusive history. Tumours, especially of the cerebellum, pressing on the vena Galeni often induce a large accumulation of fluid. In some cases the effusion appears to be due to chronic cerebral hyperæmias arising in the course of disorders of the circulation or of chronic alcoholism.

SYMPTOMS.

In many cases of congenital hydrocephalus the disease may be recognised from the birth of the child. A convulsion, even though it be a very slight seizure, occurring the first day or two of the infant's life should be regarded with suspicion. The convulsions may recur daily, or the spasms may be represented alone by strabismus, rolling of the eyes, or flexion of the thumbs on the palms. The size of the head generally attracts attention before long, although in some cases the enlargement fails to make its appearance until a considerable time has elapsed. Nutrition is early impaired, the limbs being flabby and wasted. The bowels are nearly always constipated, though diarrhoea may occur from time to time. The fontanelles gape and become distended, the anterior opening being arched. The appearance of a child suffering from congenital hydrocephalus is so characteristic that no further description need be given.

In chronic acquired hydrocephalus the symptoms present great variety. It is often very difficult to decide, when reviewing the case, what is due to the initial disease and what to the effusion. In other words, the symptoms of the chronic disorders that are known to underlie hydrocephalus—tumour, certain inflammatory affections, alcoholism, &c.—resemble the phenomena of ventricular effusion so

closely as to make it difficult to draw a distinction between them. For an accurate diagnosis the reader is referred to the sections treating of these several diseases.

When an abundant ventricular effusion supervenes upon a pronounced cerebral lesion, such as meningitis, tumour, thrombosis of the lateral sinuses, the complexion of the case is commonly subject to considerable alteration. The special senses and intellectual faculties gradually become dulled, and the patient presents an increasing listlessness and apathy. The speech becomes slow and difficult, the pupils are more or less unequal, but generally dilated, and the gait is uncertain and tottering. A general motor weakness ensues; whilst the skin reflexes and tendon jerk become considerably diminished and eventually abolished. The patient's strength gradually fails, and a tendency to relaxation of the sphincters is observed. Emotional manifestations are common; and somnolence, in some cases amounting to stupor, appears. Occasionally epileptiform convulsions occur, entirely independent of the primary disease; and a gradually increasing mental and physical helplessness, culminating in loss of consciousness, sooner or later brings the case to a close.

PROGNOSIS.

Children affected by congenital hydrocephalus very frequently die in infancy at different periods

and in different ways. A large number die under two years, though some cases survive that age by a considerable time; some even attain to manhood.

The subjects of the disease are always delicate, and are thus liable to succumb to the first serious affection that attacks them, consequently, many die from intercurrent disorders; while others gradually waste, and die from exhaustion. In some cases a sudden termination takes place in a convulsion; or laryngismus stridulus carries the child off. Some appear to recover, but they will mostly be found to be cases of rickets, with a certain increase in the normal ventricular fluid, and not true cases of congenital hydrocephalus.

TREATMENT.

West recommends salines with diuretics, and small doses of the bichloride of mercury; cold or tepid applications to the head whenever the symptoms present any degree of activity; and small doses of iodide of potassium, and the syrup of the iodide of iron with cod-liver oil "when the state is one of cachexia rather than of active cerebral disorder." Trousseau advocated Bernard's method of treatment by compression of the head. This may be carried out by strips of adhesive plaster, or, better still, by an elastic band or cap. It is not claimed that the pressure is curative, but that it simply retards the out-

pouring of the fluid. The treatment by puncture has undoubtedly been attended in some cases with success. With proper precautions, as the withdrawal of a small quantity of the fluid at each operation, and the use of a perfectly clean, small needle, the puncture can be made without any risk, provided the longitudinal sinus is avoided. It is better to remove a small quantity at a time, from four to eight drachms, and to repeat the tapping at short intervals, than to attempt to withdraw a large amount. Compression of the head should be employed along with the tapping. The puncture is best made with the finest needle of Bartleet's small aspirator, and the most suitable situation is in the coronal suture about an inch and a half from the anterior fontanelle.

CHAPTER XVI.

EPIDEMIC CEREBRO-SPINAL MENINGITIS

(*Cerebro-spinal Fever*).

IN the great majority of instances this affection, as the name implies, prevails as an epidemic, but occasionally sporadic cases are to be met with. Like tetanus and hydrophobia it is supposed to depend upon the presence of a virus in the body, and the poison, whose nature has not yet been determined, manifests a special affinity for the membranes of the brain and cord, especially the pia mater. In rapidly fatal cases hyperæmia of this membrane covering the convex and basal surfaces of the brain and posterior surface of the cord, with serous exudation beneath the arachnoid, constitute the principal morbid changes. But soon the exudation becomes purulent in character, and then it is most marked in the fissures of Sylvius, on the surface of the pons and medulla, on the upper surface of the cerebellum, and surrounding the cranial nerves; while within the spinal canal it is most abundant on the posterior surface of the cervical and lumbar enlargements. Permanent damage to the cranial nerves, spinal roots, and substance of the cord is not unfrequent. The

spleen, lungs, liver, and kidneys are often markedly congested.

ETIOLOGY.

Cerebro-spinal fever has rarely been prevalent in England and Scotland, but was common in Ireland between the years 1846 and 1850, and again between 1865 and 1867; most of the European countries have been visited by epidemics at various times.

The young are more frequently attacked than those advanced in life, and boys and young men more often than females, as may be seen from the fact that most of the recorded cases were in males between the ages of fifteen and thirty. The disease is rare after forty, but not uncommon in young children. It is generally believed not to be contagious; and though it seems to be more independent of hygienic conditions than any other epidemic affection, it occurs nevertheless where young people are congregated together, as, for example, among recruits in barracks. Some have thought that unwholesome food has originated it, especially flour made from diseased grains. It is more prevalent in cold than warm weather; and has been known to follow violent exercise.

SYMPTOMS.

As a rule the invasion is remarkably sudden, the patient being attacked in the apparent enjoyment of robust health by the following grave symptoms:—rigour, intense headache and pain in the back of the neck, vertigo, vomiting, epigastric pain, pyrexia, and marked prostration. At times the onset is more gradual; the milder form of the disease is often ushered in by prodromal symptoms such as malaise, headache, and indefinite pains, which may precede the severer ones by a few days. Very shortly—the vomiting and headache persisting—there is retraction of the head, so that the face may be turned to the head of the bed; but there is seldom marked tetanic arching of the back. In addition, the limbs become affected by spasm, either tonic or clonic; severe pain in the back radiating to the extremities and aggravated by every movement ensues; and muttering, or wild delirium sets in. In milder cases the patient may be only drowsy and restless, with slight transient attacks of delirium; and the spasms of the limbs may either be absent, or but feebly marked.

Cutaneous eruptions often make their appearance early in the course of the disease. In the very severe cases these are always petechial, but herpetic vesicles and roseola may also appear. The temperature runs an irregular course and varies very

much from time to time, in some cases it never rises above 102° F. while in others it may reach 105°. Hyperæsthesia of the skin is common. The knees are usually drawn up to the abdomen, which is retracted. There is generally constipation, and the urine may contain albumen and blood.

The following complications have often been noted:—paralysis of one or more limbs, most frequently the arms; purulent infiltration of the eye, usually the right; acute articular inflammation, which may terminate in purulent effusion into the affected joints; hæmorrhages from the nose, bowels, kidneys, &c.; and lastly, sloughing and gangrene.

DIAGNÓSIS.

The disease may be confounded with typhus, but in cerebro-spinal fever the petechial rash appears suddenly without any previous mottling of the skin, and the nervous, especially spinal, symptoms are usually more pronounced than in typhus. The fulminant cases may readily be mistaken for malignant scarlet-fever; but here again the spinal symptoms will aid in drawing a distinction, as will also the throat and rash.

PROGNOSIS.

The mortality has been found to vary much in different epidemics, but it has reached 80 per cent.

Some cases are exceedingly mild, and terminate in complete recovery in two or three weeks. Others, again, run a very short course, the patients being unable to resist the malignant vehemence of the onset succumb in a few hours or days. Some severe cases occasionally recover, but the early appearance of purpuric spots, and the occurrence of hæmorrhages must be regarded as unfavourable signs.

TREATMENT.

The attempt must be made at first to counteract by stimulating remedies any tendency to collapse should such manifest itself. To relieve pain and muscular spasm, and indeed irritability generally, opium, belladonna, bromide of potassium, and chloral may be employed. Leeches to the back of the neck and behind the ears are also useful in allaying the headache and spinal pain. Occasionally the application of ice to the head and spine affords relief. After the acute symptoms have passed away blisters may be used, and iodide of potassium and bark administered.

CHAPTER XVII.

ENCEPHALITIS—CEREBRAL ABSCESS.

INFLAMMATION of the substance of the brain dissociated from meningitis is a rare disease. It is chiefly interesting in its relation to the subsequent formation of abscess.

MORBID ANATOMY.

Encephalitis almost always occurs in the form of limited and circumscribed patches. It is never general, and is seldom diffused in the sense that the lesion is equally spread over a large tract. When the foci are at all extensive the brain tissue is infiltrated with fluid (cedematous), swollen, and studded with red spots and capillary hæmorrhages. Occasionally there is simply a more or less diffuse yellowish discolouration (*yellow œdema*) with swelling, punctate hæmorrhages, and altered consistency. The gray matter is more frequently affected than the white, which, however, may be involved in the morbid process. The cerebellum and basal ganglia (optic thalami and corpora striata) are often selected.

Encephalitis interstitialis neonatorum, the cerebral inflammation of young children, is probably the most common form of recent inflammation.

According to Virchow it is due to an increase in the cells of the neuroglia, which are enlarged and infiltrated with fatty, granular matter. The brain in this affection is enlarged, soft, sometimes much redder than normal, but sometimes of a grayish yellow colour.

Briefly, the following naked-eye appearances are generally observed in recent inflammation of the brain. The affected parts or foci of *red softening* vary very much in size. Their colour is changed; as a rule they present a reddish, sometimes a deep-red hue. Their volume is increased as the result of swelling of the neuroglia, the cut surface being raised above the surrounding parts. Throughout they are studded with minute capillary hæmorrhages; the number of points of extravasation varies in the different foci. There is no distinct line of limitation, as the neighbouring tissue, which is generally cedematous, absorbs colouring matter from the inflamed part. When the focus is small, and the inflammatory process of slight intensity, complete restoration may ensue.

More severe forms of encephalitis are apt to result in the formation of abscesses, cysts, or cicatrices attended by atrophic changes in the brain.

ETIOLOGY.

Encephalitis is most frequently the result of injury to the head. The most severe cases follow

fractures of the cranial bones, with laceration of the brain tissue; but slighter injuries, attended by contusion, also occasion an inflammatory attack, which is generally followed by chronic abscess.

It is often induced by disease (caries) of the petrous portion of the temporal bone. Erysipelas of the head and other affections likely to cause meningitis may induce inflammation of the brain substance as a secondary event.

Cerebral tumours, acute diseases (as small-pox, scarlet fever, measles), ulcerative endocarditis—by inducing multiple embolism, and alcoholic excess, may give rise to encephalitis. It probably never occurs as a spontaneous or idiopathic affection.

SYMPTOMS.

It is almost impossible to draw a clinical distinction between meningitis and encephalitis. Indeed, considering how often the latter follows injury to the head and internal otitis, and the tendency of these to induce meningitis, together with the fact that *red softening*, as a stage of circumscribed inflammation of the brain, occurs only as a transient condition, it will be found more profitable to devote attention rather to the evidences of the resulting suppuration than to the symptoms and recognition of the acute process itself.

Should an encephalitic focus occupy any part of the brain that possesses very important functions, the presence of localising symptoms, as *asphasia* and certain paralyses, may lead to its detection.

In the traumatic form after the symptoms of concussion have subsided, the patient is roused with difficulty, complains of pain in the head and giddiness, nausea and violent vomiting appear, the skin is hyperæsthetic, the gait staggering and uncertain, the pulse slow, and the temperature curve indicates fever of an irregular and remittent type.

An increase in the fever both as regards pulse and temperature, partial or general convulsions, paralysis, limited to one or more of the muscles of the eye or to those supplied by the facial, or possibly hemiplegic, along with bed-sores and deepening of the stupor, lend a greater gravity to the case.

Very chronic changes are in some cases set up in connection with traumatic meningo-encephalitis, which result in a train of more or less obscure cerebral symptoms. In others again, the symptoms disappear, and little or no permanent mischief is left. But if the patient should survive the injury for a week or two, as a rule pus makes its appearance.

ABSCESS OF THE BRAIN.

Abscess of the brain very frequently follows purulent otorrhoea when the discharge depends upon internal otitis. Meyer found it twenty-eight times in eighty-six cases of caries of the temporal bone. According to Toynbee, disease of the bony external auditory canal occasions, as a rule, lesions in the lateral sinus and cerebellum; whilst disease of the tympanic cavity gives rise to cerebral disease, and labyrinthine disease to changes in the medulla.

As any interference with the performance of the functions of the temporo-sphenoidal lobe (which are by no means well understood) is not readily made manifest, it happens not unfrequently that an acute abscess in that situation may be either entirely overlooked, or attain such a size as to cause general symptoms of compression before its presence has even been suspected. Vomiting, severe headache, often corresponding to the site of the affection, high fever, delirium, or drowsiness, and ringing in the ears and convulsions, when taken into consideration with the history of a purulent discharge from the ear which may have disappeared a short time before the commencement of the symptoms, are suggestive of abscess; but only suggestive, as they are common to several lesions. Occasionally motor tracts and nerves are

involved, when paralysis of a more or less extensive character will be observed. But in the great majority of cases the symptoms are those of a rapidly developed and increasing cerebral compression, ending soon in deep coma, and accompanied by convulsions.

According to Huguenin some abscesses dependent upon ear disease are not preceded by a stage of acute focal inflammation or *red softening*, but appear as collections of pus from the commencement.

In the course of pyæmic cases abscesses are occasionally developed in the brain. The formation of pus here as elsewhere may be preceded by a rigour. Headache, giddiness, somnolence, delirium, and convulsions are the usual cerebral symptoms in such cases.

CHRONIC ABSCESS OF THE BRAIN.

Chronic abscesses result from several conditions. They are apt to follow disease of the internal ear, or injuries to the head, or they may depend upon irritation set up about a blood-clot. When the abscess has existed for some time the pus is usually found within a tolerably thick capsule, which may be readily enucleated.

In many cases the symptoms are more or less in abeyance for a considerable period, during which the disease is in fact almost latent, and only made active, it may be, in response to a further provocative in the direction of a blow, undue excitement, &c.

Headache, at times very slight, and at times severe and paroxysmal, is a feature of the majority of cases from the commencement. Giddiness is rarely entirely absent. Vomiting may appear occasionally, but the patient is often free from it during the obscure or latent period of the disease, whilst it commonly accompanies the headache when it returns in paroxysms. These paroxysmal attacks are often followed by weeks marked by complete immunity from all cerebral symptoms. Convulsions sometimes appear, though rarely, in this stage. Should localising symptoms of chronic abscess be present, no distinction can be drawn between them and those resulting from any other local disease.

After the existence for some time of the somewhat obscure symptoms which occur in the so-called *latent stage* of chronic abscess, the case undergoes a complete change. This generally occurs rapidly, and is characterised by general inflammatory symptoms, vomiting, severe headache, giddiness, loss of consciousness, delirium and coma, ending soon in death. In some case

these symptoms stop short of the fatal result, and a temporary recovery ensues, soon, however, to be followed by a second, and probably a fatal, relapse. These alarming symptoms constituting the terminal period of chronic abscess differ very considerably in individual cases; their precise nature is more or less dependent upon the particular change which has induced them.

Sometimes the symptoms arise from rupture of the abscess on the surface of the brain, when an acute and rapidly fatal meningitis supervenes. Again—and this is the most usual event—death occurs as the result of œdema of the brain, caused no doubt by a sudden increase of the abscess. Or the abscess may perforate the walls and enter the cavity of the lateral ventricle, an event attended by convulsions, paralysis, loss of consciousness, delirium, and death. The convulsions are usually bilateral, and the facial muscles are generally prominently affected.

Some patients make no complaint at all until the terminal period arrives, the abscess having formed independently of initial encephalitis. Such a history is most frequently elicited when ear disease is the cause of the abscess; indeed, an encapsulated abscess of the temporo-sphenoidal lobe may remain quiescent for a long time. The clinical records abound in cases of the sort.

Chronic abscess associated with thrombosis of

the lateral sinus is often very difficult to recognise, as the symptoms of the former affection may be masked by those of the latter. In some cases the combined maladies give rise to very few symptoms; in others it is easy to recognise a gross but indefinite cerebral disease. But headache, giddiness, vomiting, and perhaps rigours, added to the signs of thrombosis (unilateral swelling of the lids and injection of the conjunctiva, one-sided œdema of the face and mastoid region, with thrombosis of the internal jugular vein and emptiness of the external jugular), should suggest co-existing abscess and thrombosis.

DIAGNOSIS.

Abscess of the brain is regarded by practical physicians as a very difficult disorder to diagnose. The symptoms bear more resemblance to tumour than to any other affection. The history of otorrhœa is certainly suggestive of abscess, and injury stands in a prominent causal relation to both; in my experience it is more common to meet with the history of accident in tumour of the brain than not. When the injury has been followed by symptoms of encephalitis, and then by an interval of two or three months during which time obscure symptoms are present, to be succeeded by a more active period, the indication

is in favour of abscess. On the other hand, signs of a more definite character (headache, vomiting, giddiness, &c.), spread over a longer period, say, a year or year and a half after a blow, point to tumour. Well-marked double optic neuritis, especially if the disc is much swollen and many hæmorrhages are present, discovered after the existence for a considerable time of headache and vomiting, should be regarded as favourable to the diagnosis of tumour. Motor paralyzes and sensory disturbances of a marked character are much more common in tumour than in abscess.

It has already been remarked that meningitis can seldom be distinguished from encephalitis. Bilateral phenomena in the direction of paralysis and convulsions, along with very high fever and violent headache, are suggestive of meningitis; but more limited convulsions and paralysis attended by less fever point to the presence of encephalitic foci alone.

PROGNOSIS.

Care must be taken not to allow apparent recovery from an attack attended by alarming symptoms in the course of an abscess to interfere with the exceedingly grave prognosis that should be made in all such cases, for they are always liable to a relapse.

Traumatic encephalitis or meningo-encephalitis usually ends fatally, and recovery only takes place when the suppuration is not diffuse, and the pus can be discharged through the wound. In chronic traumatic abscess death usually occurs sooner or later; in some few cases, however, trephining has been successfully performed, and in a few others the abscess has discharged spontaneously with a fortunate result.

The prognosis of abscess depending upon ear disease is also very unfavourable, though some cases *appear* to recover by the pus discharging outside.

TREATMENT.

An attempt may be made to combat the initial encephalitis occurring after injury by cold applications, rest, and purgatives. Leeches should be applied behind the ear, and afterwards warm poultices, and the auditory canal should be frequently washed out with warm unirritating injections, when symptoms of encephalitis develop after otorrhœa. Sometimes purulent matter passes from the tympanum into the mastoid cells, an occurrence fraught with danger, and one which will sometimes be revealed by redness and swelling over the mastoid process, when an incision should always be made down to the bone to provide an exit for the pus. It is clearly beyond my pro-

vince to discuss the indications for, and methods of practising, operative interference in cases of abscess of the brain further than to say that trephining the mastoid cells is always a wise procedure in cases depending on ear disease. The physician has to diagnose and localise the abscess, but the remainder must rest with the surgeon.

CHAPTER XVIII.

SYPHILIS OF THE BRAIN.

THE manifestations of syphilis in the brain may be said to belong to the later secondary or to the tertiary stages of the disease, although in some unusual cases it has been known to develop a few months after the infection. In others again, the specific lesion has been postponed for as many as thirty years. It shows itself with the greatest frequency during middle life.

MORBID ANATOMY.

Syphilitic lesions which give rise to cerebral symptoms may be situated in the cranial bones, in the membranes, in the cranial nerves, in the cerebral arteries, or in the brain substance.

Under the influence of syphilis any one of the various kinds of inflammation to which bone is liable may be produced; thus we meet with exostoses, periostitis, and caries—conditions which produce pressure upon the brain and nerves, and set up in them secondary inflammations.

The membranes may be the seat either of distinct specific tumours (gummata), or of inflammation and thickening. Syphilis of the dura mater is generally seen in the form of dense

thickenings, with adhesions to the bones and subjacent membranes. Osteophytes may be developed in the indurated structure. In the pia mater the inflammation is generally chronic, and very often limited in extent. This circumscribed chronic inflammation is marked by thickening and adhesions, and in some cases gummatous exudation is spread over the surface of the brain in the membrane. The connective tissue elements which result from the inflammation contract, and exercise pressure upon the surface of the convolutions and nerves, causing more or less atrophy.

Syphilitic gummata are very often difficult to distinguish from gliomata on the one hand and scrofulous tumours (tubercular disease) on the other. The gummous tumour is generally connected with the surface of the brain, and, in consequence, causes the membranes to be adherent. It is usually irregular in form, and of a firm consistence, with a pinkish-purple colour, but in parts yellow owing to caseous degeneration. In some cases the syphilitic tumour appears translucent, or gelatinous, in parts pinkish-gray, and in parts yellow. It is very often single, and more or less diffused at the circumference, the margin being very seldom distinctly circumscribed. The largest gummata are found in connection with the pia mater covering the cerebellar peduncles.

A more diffused syphilitic change occurs in the

form of so-called *sclerosis*, a variety of disease that is seldom met with in the brain apart from cord and peripheral disease.

The arterial walls are often very much thickened, and the lumen of the vessel may be obliterated, as the result of syphilitic infiltration. The deposit takes place chiefly in the adventitia of the artery, or it may occur between the intima and the endothelium.

The effect of this peri-arteritis or endarteritis upon the brain is necessarily to diminish its blood-supply, an event which may be attended by necrotic softening.

The fissure of Sylvius and the cortical substance in the region of distribution of the middle and anterior cerebral arteries are favourite seats for syphilitic deposit. When a *gumma* develops within the fissure it usually invades the middle cerebral artery, and hemiplegia with or without aphasia occurs.

The cranial nerves may be affected either by being implicated in thickened membranes, or by compression by gummata, or else by the trunk of the nerve itself becoming infiltrated. In most cases the nerves are implicated at their superficial origins before they become covered by the dura mater.

Virchow has called attention to the fact that the parts most frequently affected are those most

exposed to injury, making it probable that injuries stand, in some measure, in an etiological relation to gummata—a view which my own observation has certainly led me to endorse.

In certain rare cases of congenital syphilis the brain is attacked during infancy.

SYMPTOMS.

Headache is one of the earliest manifestations; as an initial sign it is seldom absent no matter what form the disease may subsequently assume. The pain is at first slight and intermittent, but later on severe, and characterised by nocturnal exacerbations. It may be regarded as a premonitory symptom, and has been ascribed by Heubner, in this connection, to extra-cranial inflammation. It is usually more or less localised, and is wont to become intensified on pressure. Giddiness, insomnia, neuralgic pains, loss of memory, and irritability of temper may also be regarded as premonitory symptoms; sleeplessness being especially frequent and the most important of the group.

As the disease develops, after the premonitory stage other symptoms of a more pronounced character make their appearance. They present great variety, and are determined by the nature and situation of the lesion. In some cases a fit, either epileptic or apoplectiform, marks the onset, calling

attention to the presence of a serious organic lesion. In others, the decisive symptoms creep on comparatively slowly, and the case runs a chronic course.

Next to headache motor disturbances are most frequently met with. Paralysis may develop suddenly after a fit, when it will generally be found to be extensive; hemiplegia is said to occur in about one-third of the cases of cerebral syphilis.

With tolerable frequency a limited paralysis occurs suddenly. A patient, whilst comparatively well, all at once squints, or sees double, or his eyelid droops, or his face becomes twisted. Both kinds of paralysis, the extensive and the limited, may, however, come on slowly. The third cranial nerve is the one most frequently attacked, and as a rule ptosis appears before squinting. The seventh is also very often affected, but the hypoglossal is almost never the seat of syphilitic disease as an isolated lesion.

In addition to the various motor derangements of a paralytic nature, syphilis occasionally induces muscular spasms which may be limited to a single muscle, or extend to a group; and also other irritative phenomena, of a sensory kind, such as neuralgia of the branches of the fifth nerve and in the occipital region. At a later period the parts supplied by the trigeminus may become anæsthetic, but seldom to an exaggerated degree.

Any of the special senses may be attacked, and the visual oftener than the others. Ordinarily the eye is affected by optic atrophy from intracranial disease of the optic nerve, choroiditis, or perhaps by a slight diplopia. Most frequently the impairment of the other special senses depends upon paralysis of the fifth.

In many cases psychical disturbances form a prominent part of the clinical picture. At times the predominant condition is one of apathy and indifference, amounting, it may be, to somnolence. In other cases fretfulness and irritability of temper prevail. The memory is generally weak, and before death a state of helpless imbecility may be reached. Some cases pursue a course very similar to general paralysis of the insane.

When the specific deposit assumes the form of a distinct tumour (gumma), it gives rise to the symptoms which may be said in general terms to be characteristic of cerebral tumours, namely, headache, vomiting, and double optic neuritis, as well as localising symptoms the nature of which is decided by the situation of the tumour in the brain.

DIAGNOSIS.

A history of syphilis and present indications of the disease should be sought for in every case of brain and cord affection. It must be recollected that in a large proportion of cases syphilis appears

in the brain after the commoner evidences of its earlier manifestations have vanished; hence the greater necessity for making a diligent search after old-standing signs of the specific taint. In suspicious cases every remote corner of the body, so to speak, where the disease is known to leave traces of its former ravages should be carefully examined. In making a diagnosis the aid of the ophthalmoscope will be valuable in revealing old choroiditis which, should it be present, will furnish at once the required information.

Rapidly developed paralysis of the cranial nerves, in the absence of the symptoms of intracranial tumour, points to syphilitic disease, and the presumption is further strengthened on witnessing the disappearance of these paralyses, especially if at the same time epileptiform convulsions supervene.

The constitutional disturbances attending an attack of syphilitic meningitis are rarely pronounced: this fact, when taken into consideration along with the excruciating headache, intensified on pressure, is a significant feature in the diagnosis.

In distinguishing syphilitic occlusion of a vessel with consequent softening from ordinary thrombosis, youth, absence of general atheromatous disease, and the co-existence of peripheral lesions of the cranial nerves point to the former condition.

It is often very difficult to decide between syphilitic and other intracranial tumours, but the character of the pain may furnish a clue. The headache is generally more severe when the lesion is a gumma, as the membranes are nearly always markedly affected; whereas in the case of the other varieties of tumour the lesion may be so situated as only to affect the membranes in an indirect way.

PROGNOSIS.

Syphilitic disease of the brain very frequently ends fatally. Perhaps no cerebral disease is more difficult to prognose. In few cases can a hopeful opinion be formed, and it often happens that just as some slight improvement seems to justify a more favourable view of the case, a sudden and unlooked-for complication arises to upset the anticipation.

Syphilitic arterial disease is probably the most serious of all the forms the affection assumes in the brain.

TREATMENT.

Anti-syphilitic remedies should be employed very early, as the disease will obstinately resist them when it has gained a firm footing. At the same time it must be borne in mind that diseases which owe their origin to syphilis yield

more readily to appropriate treatment than any other variety of organic cerebral disorder.

Mercury will generally be called for either alone, or, what is better, in combination with iodide of potassium. The administration of these drugs should be kept up for a considerable time, care being taken not to induce salivation. The bichloride and the iodide of mercury are probably the best preparations, and sometimes the combination of gray powder and quinine is very efficacious.

CHAPTER XIX.

INTRACRANIAL TUMOURS.

MORBID ANATOMY.

THE *gliomata* or *glio-sarcomata*, which constitute the most important class of brain tumours, are formed by proliferation of the cement substance or neuroglia. They vary very much in size and shape; some are no larger than cherry-stones, whilst others are half the size of the hemisphere. The cut surface of the tumour usually presents a pink or reddish-purple colour with yellow patches resembling cheesy matter, the result of retrogressive changes; but in some instances it is very pale, and difficult to distinguish from the surrounding brain substance. The *gliomata* are often extremely vascular, and as many of them are of soft consistency hæmorrhagic effusions into their substance are by no means uncommon. They are most frequently found in the hemispheres. Their development is generally slow, and they are seldom circumscribed and clearly defined, but show a tendency to blend with the neighbouring brain matter.

The *scrofulous* or *tubercular* tumours are also frequently met with, and appear generally as firm

nodules of a yellow colour, with a thin reddish vascular cortex. They may be any size up to a large hen's egg, and are commonly spherical in form, and tolerably distinct from the parts around. Small cavities are wont to form in the centre of the growth from softening of the tissue. They may be seated in the cerebrum, but are more frequently found in the cerebellum.

Syphilitic tumours have already been described (p. 165). They are nearly always connected with the cortex, and are distinguished on section by the gelatinous appearance they usually present. Indeed, the typical cerebral gumma may be said to resemble an uncooked piece of a white fish, with patches of yellow-coloured tissue scattered through it.

Cancerous tumours occur somewhat rarely in the brain, although the opposite statement is made by some writers on the subject. Of twelve cases of brain tumour that have come under my observation in which the diagnosis was verified by post-mortem examination, only two were of a cancerous nature. The so-called *fungus duræ matris* is one of the commonest varieties of this kind of new-growth.* It may spring from either surface, and frequently perforates the skull when it appears externally in the form of a fungating mass. *Endotheliomata* or "nested sarcomata" are also to be met with growing from the dura

* These tumours, though malignant, really belong to the sarcomas.

mater; they resemble, on account of the concentric arrangement of the cells, the ordinary epitheliomatous tumour. But cancer (?) may develop in the brain substance also, the medulla oblongata and crura being rarely affected.

The above are the commonest forms of intracranial new-growths; but, in addition, the following tumours may make their appearance.

Different varieties of *sarcomata*.

Psammomata, or sand tumours, new-growths which contain granules of crystalline matter, but which must be distinguished from other tumours that have undergone calcareous degeneration. They usually grow from the dura mater at the base, and are white and round like a cherry stone.

Lipomatous growths have occasionally been found within the ventricles.

Myxomata are sometimes found, but tumours of this nature are generally gliomata that have undergone myxomatous degeneration.

A peculiar form of tumour known as *cholesteatoma* which appears to spring from the pia mater, is sometimes met with at the base. It presents a pearl-like lustre, and may be the size of a cherry, and is generally the result of the coalition of a number of minute bodies.

The *pineal gland* may become hypertrophied, and develop into a tolerably large tumour.

Two forms of encysted *parasites* appear in the

brain, the *Cysticercus cellulosæ*, and the *Echinococcus hominis*.

Cysticerci are usually found in the substance of the hemispheres, though they may be seen on the surface of the brain. It is most exceptional to find them free, but occasionally they are observed in the ventricles. The meninges, especially the pia mater, often contain them.

The cerebral cysticercus is usually inclosed in a very soft spherical envelope in which the animal is seen with the naked eye as a small white tubercle; under the microscope, the neck of the parasite becomes visible with the circle of characteristic hooklets curved inwardly (Rosenthal).

Echinococci attain much larger dimensions in the brain than the cysticerci, but they occur much less frequently. These cysts are composed of firm external membrane which incloses the parasites. On the internal surface of this membrane, which usually contains a clear liquid, are found small bodies the size of a millet seed, with the characteristic ring of hooklets. The cysts are most commonly situated in the hemispheres, but may be found in the cerebellum and elsewhere.

Aneurisms occur within the cranial cavity but rarely, of course excepting the small miliary

aneurisms which are so often the cause of cerebral hæmorrhage. As a rule they are situated at the base, though I recently met with an aneurism the size of a cherry springing from one of the middle branches of the right anterior cerebral artery, which occupied the right gyrus fornicatus about its middle portion.

ETIOLOGY.

Cerebral tumours occur more frequently in men than women. The appearance of scrofulous and syphilitic growths implies previous strumous and syphilitic disease in the patient. The hereditary character of cancer ought to be remembered in the diagnosis of a doubtful case. Cerebral cancer is generally primary, though some authorities* hold the contrary view.

Scrofulous tumours of the brain are usually associated with tubercle elsewhere, but at more than one post-mortem examination I have met with a scrofulous intracranial growth without any disease of a like nature in any other part of the body. Cancerous tumours are more common in advanced life; scrofulous growths, on the other hand, generally appear in young people; while syphilitic depend on the period of the primary infection. Various kinds of tumours have been

* Orth : "Diagnosis in Pathological Anatomy," p. 80.

traced to injury to the head, and I must reassert the opinion I have already given that traumatism must be held responsible for a large proportion of intracranial new-growths, notwithstanding Jaccoud's opinion that the apparent relation of injury to tumour is purely fortuitous.

SYMPTOMS.

In general terms the features which lead to the detection of a cerebral tumour arise from increased intracranial pressure, and derangement of the functions of the affected part will enable the observer to localise the growth. The early appearance of headache in the great majority of cases may be expected, and when the membranes are not directly implicated in the growth, the pain, which is often paroxysmal, and may be most intense, arises commonly from stretching of the dura mater. Not unfrequently an interval of days or weeks marked by perfect freedom from pain may intervene between the paroxysms. In other cases, again, the headache is almost constant, usually when the membranes are implicated. There are on record a few rare cases in which this symptom was never complained of, but in those which have come under my own observation the headache was present at one time or other.

Vomiting is another most important symptom.

Though often occurring with the headache and resembling it in its paroxysmal character, it is more frequently absent, and seldom makes its appearance so early in the case. It is undoubtedly a reflex act arising out of the probable connection between the vagal nucleus and the long sensory root of the fifth in the medulla, the latter of which supplies the dura mater with sensation.

Giddiness is most frequently present, and commonly in a marked degree. It is very often the first sign of a departure from health. Indeed, arising as it does in a great proportion of cases from an alteration in the circulation of the brain, it is easy to understand that an increase in the intracranial pressure will readily give rise to it.

Mental disturbances, such as loss of memory, irritability of temper, &c., are of exceedingly common occurrence, though this is denied by Andral, Durand-Fardel, and others.

Optic Neuritis must be regarded as a sign of paramount importance far exceeding in diagnostic value the symptoms already named. It is impossible to lay too much stress upon this condition of the optic disc; for symptoms that before were obscure and apparently meaningless, can at once be awarded their true significance on the discovery of double optic neuritis. In other words, headache and giddiness, with perhaps attacks of

vomiting, may appear by themselves indefinite symptoms, but when associated with double optic neuritis they leave little doubt, in the absence of lead poisoning and kidney disease, that the case is one of cerebral tumour. Unfortunately we have not the guidance of the swollen disc either in every case or in every stage of the disease; but in a very large proportion of cases, if it be not present early it will appear later on. It may be deferred for a very long time; thus, in a patient recently under my care who suffered from paralysis of all four extremities, the result of a tumour in the medulla which had been apparently growing for more than ten months, the optic neuritis only began to show itself ten days before death.* So late a postponement is exceedingly rare. It is important to notice that patients with even intense double optic neuritis can, as a rule, see perfectly well; this fact, which has been denied by some ophthalmologists, has been made so plain by Dr. Hughlings-Jackson that it need only be casually referred to here.

Motor Disturbances.—Epileptiform attacks are of rather frequent occurrence. They commonly resemble closely the convulsions of idiopathic epilepsy, from which they may be often distinguished by their onset and development. In epileptiform attacks caused by tumour the commencement is

* See Case III., p. 187.

usually local, and the convulsions confined to one side of the body. Again, when an aura is present in organic disease, it is usually a visual or auditory phenomenon. Consciousness may or may not be lost in convulsive attacks attendant on a tumour, and in many cases is lost only after the fit has well commenced. The motor phenomena of irritation are often simply spasms or cramps which may be purely local, as in the case of clonic spasms of the muscles of one side of the face. In one of my cases when the spasm came on, the fingers of the right hand were slowly clenched, and the forearm was flexed on the arm. After a seizure the convulsed muscles are generally more or less paralysed, and this post-epileptic paralysis is wont to last much longer than in the case of functional epilepsy.

The greatest possible variety of paralyses occurs in cases of tumour, from paresis of a single ocular muscle giving rise to diplopia alone to complete paralysis of all four extremities. Hemiplegia is probably the commonest form, though, in some cases, alternating paralysis makes its appearance; but of course the precise nature of the paralysis will depend on the situation of the lesion.

Disturbances of sensibility are also common events in cerebral tumours; they are either irritative or paralytic. The irritation is shown

by hyperæsthesia, or by neuralgia; the occurrence of the latter in all the divisions of the fifth nerve at once being suggestive of a tumour. It is not unusual to meet with anæsthesia of the parts supplied by the branches affected with neuralgia. In such a case compression of the trunk of the nerve by a new-growth should be suspected.

Numbness, "needles and pins," and other subjective symptoms, are often complained of. The whole of one side of the body may be, but seldom is, completely anæsthetic, more or less sensibility being generally left in the affected part. However, a progressive hemi-anæsthesia in a male patient points very plainly to tumour. No doubt the ordinary sensibility frequently remains unaffected; and, in some cases the impairment of mental vigour so often noted may produce an imitation of anæsthesia.

The special senses are frequently more or less impaired, and the eye is the organ most liable to suffer. Diplopia is very often complained of, and may for a considerable time be the only symptom of deranged cerebral function. Amblyopia is seldom marked, unless the optic nerve is atrophied or the yellow spot is occupied by hæmorrhages. But after the optic neuritis has existed for some time the nerve degenerates, when it may be seen with the ophthalmoscope becoming gradually

whiter and whiter, the sight becoming correspondingly impaired.

The sense of smell is, I think, more often affected than most writers on the subject are prepared to allow. At the same time it must be confessed that, except in the case of tumours of the frontal lobes, paralysis of the fifth is undoubtedly the usual cause of the impaired olfactory sense.

Taste is not often affected, and it is very rarely completely lost.

The sense of hearing is more frequently disturbed. The disturbances vary from a confused noise in the head to complete deafness arising from compression of the trunk of the portio mollis.

As the growth advances the headache and vomiting become more constant, the patient emaciates, his mental condition grows more enfeebled, his sight fails entirely, and the paralysed extremities become rigid. Death is often due to cerebral œdema; or effusion into the ventricles may hasten the termination of the case.

The following very brief reference to three interesting cases of intracranial tumour will serve to illustrate the subject. The first is an example of a glioma, the second of a syphilitic growth, and the third of a tubercular tumour.

- I. *Glioma of the corpora quadrigemina affecting the adjacent part of the cerebellum and the posterior portion of the right optic thalamus (fig. 23).*

The patient, a married woman, aged 38, was admitted into the Newcastle-on-Tyne Infirmary

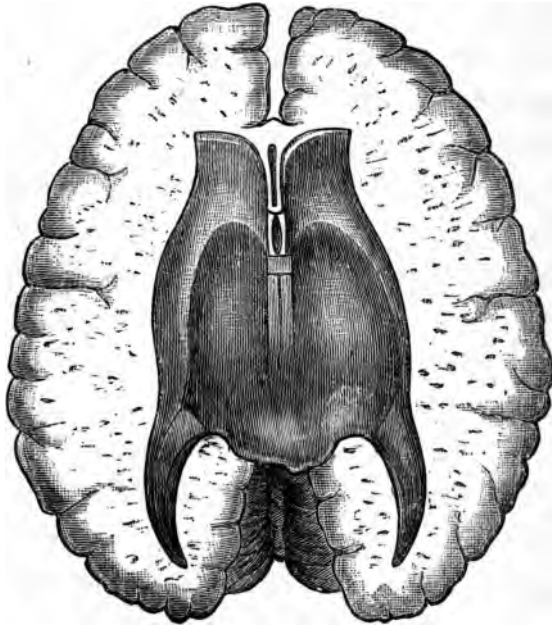


FIG. 23.

on the 18th of July, 1881. She stated that she had been out of health for about twelve months. At first double vision was complained of, and subsequently headache and giddiness, with occasional attacks of vomiting. At the date of admission

the principal features were headache, vertigo, diplopia (due to slight convergent squint), a staggering gait, some vomiting, and double optic neuritis. Subsequently nystagmus was observed, and a greater degree of blindness developed than could be explained by the ophthalmoscopic appearances, even though intense swelling of the papillæ and hæmorrhages were present. The inco-ordination became very marked, so that she could not even sit up in bed. At a later period, all four extremities became more or less paralysed, but especially the left arm and leg. Bed-sores formed, and she died on the 12th of November, 1881.

II. *Syphilitic gumma growing from the pia mater covering the right cerebellar peduncles, and compressing the cerebellum, medulla, and pons (fig. 24).*

The patient, a married woman, aged 27, was admitted into hospital, November 16th, 1881, complaining of occipital and frontal headache, vomiting, and a staggering gait. She had suffered from syphilis. The headache was of seven months', and the reeling gait of three months', duration. Nystagmus, a tottering, staggering gait, headache (principally in the back of the head), right facial neuralgia, vomiting, and double optic neuritis were

the leading points of the case on admission. In time the erect posture could not be maintained, owing to her inability to co-ordinate: there was always a tendency to fall to the right side.



FIG. 24.

Anæsthesia of the right side of the face and tongue made its appearance, with incomplete right facial palsy; and motor paralysis of both arms and legs developed. The optic neuritis gave place to atrophy, and blindness ensued. The patient died five months after her admission into hospital.

III. *Scrofulous or tubercular tumour of the medulla oblongata* (fig. 25).

The patient was a sailor, aged 32, and was admitted into hospital on the 2nd of February, 1882, complaining of paralysis of both arms and

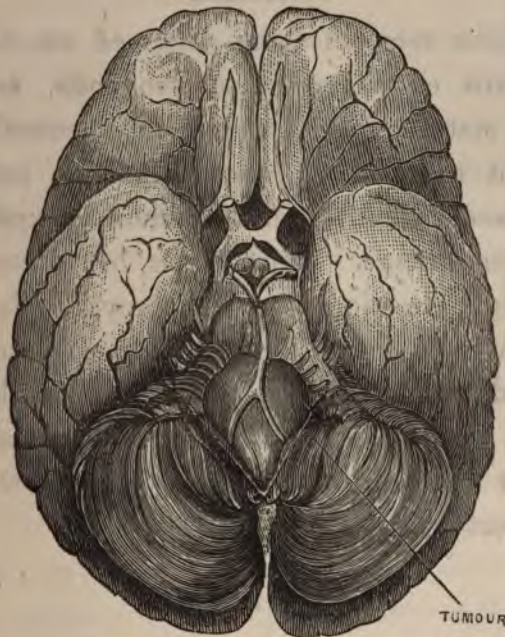


FIG. 25.

legs, headache, vomiting, difficulty in swallowing (dysphagia), difficulty in speaking (dysarthria), and inability to protrude the tongue. He stated that his illness commenced about five months before admission with debility and headache. A

month subsequently the left leg became weak and heavy, and before long the paralysis extended to the other extremities, eventually involving the tongue. Optic neuritis only developed ten days before the patient's death, which occurred three and a half months after his admission.

PROGNOSIS.

Syphilitic tumours alone admit of anything of the nature of a favourable prognosis, and, as already stated, gummata are only amenable to treatment early in the case. Patients suffering from vascular gliomata often die most unexpectedly from hæmorrhage. Some cases of tumour are exceedingly chronic, and may improve so far that a patient will leave off attending at the out-patient department of an hospital under the impression that he is cured; but no medical man who has witnessed the progress of a few cases of the sort will share in this fallacy.

TREATMENT.

In the absence of a syphilitic taint treatment can be of little avail. Bromide of potassium, chloral, especially croton chloral, and opium are of service in relieving pain, but all that can be done is simply in the direction of mitigating the sufferings of the patient.

CHAPTER XX.

GENERAL PARALYSIS OF THE INSANE.

ALTHOUGH this disease has been clearly recognised since its description by Bayle in 1822, and has since that time received an immense amount of attention from physicians specially interested in mental diseases, it is yet all but unknown to the profession generally, and is seldom mentioned by teachers of general medicine in their lectures. Consequently, students finish their professional education in complete ignorance of such a disease. As a natural result it is seldom recognised in its earliest stages; and when at last the family doctor does see that his patient's mind is disordered, the case is usually put down as one of "softening of the brain," unless, indeed, acute mental symptoms appear, and then it is sent to an asylum as one of acute mania. For the patient this is a most unfortunate circumstance, as he runs a great chance of forfeiting his previous character and good name. For example, before the mental symptoms are sufficiently developed to attract attention, he may have ruined his business, covered himself and family with disgrace through his shocking immorality, and may even have got entangled in the clutches

of the law. These most distressing cases are by no means rare; and it is therefore the duty of every practitioner to look minutely for the earliest physical symptoms of general paralysis whenever he finds that the habits of a previously careful and respectable man have changed rapidly to the contrary.

MORBID ANATOMY.

At the outset we may observe that there is great hypertrophy of the connective tissue discernible throughout the brain and cord. This fact explains most of the other changes, and really furnishes a clue to the pathology of the disease. General paralysis is a genuine example of cirrhosis of nerve structure. The microscopic changes have been conveniently arranged under three heads, as affecting—1, the vessels; 2, the neuroglia; 3, the nervous tissues proper.

The following paragraph from Dr. Mickle's book* gives an epitome of Voisin's views as to the changes in the vessels, and we reproduce it here as being sufficiently full and accurate:—

“Writing in 1875, with reference to dysphasia in general paralysis, Aug. Voisin mentioned in certain cases ‘hyperæmia, infiltration with blastema, with exudation and recent embryoplastic

* Mickle: “General Paralysis of the Insane.”

production, and softening, of the cortical layer of the anterior lobes of the brain.' The vessels present in their walls, and in the spaces by which they are surrounded, an abundance of embryoplastic nuclei in very close rows; the nervous substance itself contains a numberless amount of these nuclei. They may also be seen 'in the course of the nervous fibres which pass from the cortical substance of the anterior convolutions to the medulla oblongata through the white cerebral substance, the corpora striata, and the pons Varolii. In the course of these fibres and in their interior you meet with an enormous quantity of embryoplastic nuclei, which are especially abundant in the vascular sheaths in the perivascular spaces, and which thence certainly have invaded the nervous substance; and you meet, also, with masses of hæmatosine, of hæmatine, in yellow or colourless crystals, or effusions of blood-corpuscles more or less old.' In his recent work Voisin describes in the brain cortex, endarteritis, evidenced by nuclear bodies, round or oval, more or less packed, and forming encircling chaplets or muff-like surroundings of the vessels;—perivascular blastemic effusion;—effusion of blood-globules around the vessels, and hæmatine crystals in and about their walls, with hæmatosine granulations in the perivascular lymph-space, which last is often increased in

width;—encircling chaplets of fusiform bodies around the vessels, hæmorrhages of the external coat of the vessels, and hence compression of the vessel from without;—ampullary dilatations or miliary aneurisms, or compression or obliteration of vessels by the organisation of fibrillary tissue from the cellules lining their walls;—and colloid degeneration.”

The changes in the neuroglia are sufficiently referred to in the preceding paragraph. Although Westphal and others have strenuously denied their existence, there appears to be no reasonable doubt that they are very marked, though they may vary in different portions of the nervous system, and in different cases; indeed, as already stated, they are the first to arrest attention.

The connective tissue developed in the cerebral cortex and elsewhere by pressing upon the nerve cells and their fibres causes them to become atrophied. The cells are altered in appearance,—become filled with pigment or otherwise changed, and their branches atrophied or lost.

It is necessary to describe the naked-eyed appearance of the brain and cord when removed from the body.

In an average case there is at once evident shrinking or atrophy of the organs. In the brain this may cause a loss of weight of 3-5 oz., but it

is difficult to state exactly its amount in the cord. The arachnoid is thickened and presents an opaque, leathery appearance, and the pia mater is loaded with fluid. These changes are most marked in the frontal and parietal regions; indeed the membranes covering the occipital and temporo-sphenoidal lobes may appear almost normal. The membranes are adherent here and there to the tips of the subjacent convolutions; and an attempt to strip them will tear away some of the gray matter, leaving a roughened surface. This is very characteristic of general paralysis. The brain-substance generally may be what is called watery, or, on the other hand, of normal, or firm consistence. Where the atrophy is most marked, the convolutions may be distinctly hardened and sclerosed. On section, the gray substance is seen to be atrophied, and its tint changed. The lateral ventricles contain an excess of fluid, and the lining membrane is thickened and roughened, presenting a finely granular appearance. This also is highly characteristic, and is due to chronic inflammation of the ependyma. As a rule it is even more marked in the fourth ventricle.

ETIOLOGY.

A few words as to the causes of general paralysis. Although we cannot believe that the disease was

a new one when it first attracted Bayle's attention, it is no doubt much more common in the present day. It would appear as if the struggle for existence, so severe in modern times, predisposes to, or actually causes, this fatal disease. Yet this supposition does not satisfactorily explain all the circumstances of the case. In England it is more common than in Scotland, whilst in the latter country it is more frequently met with than in Ireland where it is a comparatively rare disease, even in the large towns. Yet when Scotchmen and Irishmen come to England or go abroad, they suffer from general paralysis frequently enough. Although it is quite true that a "fast" life tends greatly to its development, still immorality and drunkenness do not appear to be of themselves sufficient to originate it. We know that these vices are common enough in the Scottish Highlands, yet it would appear as if a Highlander could indulge with impunity, so long as he remains in his native glen. Should he, however, frequent the centres of civilisation, and there continue his evil habits, he is as liable as any other to become a victim. This fact has given rise to the belief that syphilis has much to do with its development, and there is probably some truth in the suggestion. Some forty years ago it was a very rare disease in Scotland amongst men, and unheard of in women; nowadays it is common enough in both sexes, especially in large towns.

When we compare the conditions of life of the inhabitants of Hull, Glasgow, and Belfast, we cannot discover any reason why general paralysis should be so common in the first, so seldom seen in the last, and why Glasgow should hold a position between the two. It is a remarkable fact that the members of the Society of Friends should be almost entirely free from the disease. Doubtless this immunity may be traced to their comparative virtue and self-control, even amid the cares and excitements of life.

Sex powerfully predisposes to general paralysis; men being at least five times more liable to it than women.

The disease is one of mature life. It generally occurs between the ages of 35 and 50, but it has been seen in a lad of seventeen. It is specially fatal to sanguine, ambitious, pushing men.

To the chief exciting causes, alcoholic and, probably, sexual excesses, may be added business and other anxieties in their widest sense, cranial injuries, and sunstroke.

SYMPTOMS.

The clinical aspects of general paralysis are so varied that it has been suggested that there are included in one class a variety of diseases which might in time be differentiated. Be this as it may, it is quite as likely that the phenomena can be

explained by the morbid changes in the nervous system varying in their site and intensity. Although it may with perfect accuracy be said that in every case the whole nervous system is involved, it is also true that in each one the changes are more marked in some special locality. In a typical case the changes affect about equally both brain and cord; but we often find the changes more marked in the brain than in the cord, and *vice versa*. Again, whilst as a rule the pathological changes are most marked in the frontal and parietal regions of the brain, they may affect one hemisphere more than another, and one convolution or part of one more than another. We thus see that the varieties of symptoms are capable of simple and rational explanation.

In studying the clinical features of the disease, it must be remembered that there are two classes of symptoms, the physical and the mental. Speaking generally, these proceed together, and diminish or increase at the same time and in the same degree, though cases are seen often enough when one class improves much more than the other. Thus a general paralytic may improve so much for a time that the mental symptoms may entirely disappear, and the only indication of disease may be a slight tremor of the tongue, or of the muscles of the angles of the mouth when the patient speaks.

Long and fruitless discussions have taken place as

to whether the physical or mental symptoms appear first. It is seldom that cases are seen early enough for this point to be decided. At first the physical and mental symptoms are so slight that they are apt to escape any but the most minute examination. For example, it is scarcely to be expected that slight *inequality of pupils* will at once attract the attention of a household; or that *forgetfulness* or *irritability* are likely to suggest an approaching attack of insanity. Once the illness is thoroughly developed, and the relatives begin to fix the earliest date of mental alteration, circumstances which had previously passed unnoticed, or excited transient surprise, are easily recalled. It should be remembered that the first symptom to attract attention is not necessarily the first to arise. Thus it happens that we are rarely in a position to say whether the physical or mental symptoms appeared first; and for practical purposes it is of no moment.

It is usual to divide the course of general paralysis into three stages or periods.

The first is that of incubation. Of the earliest or prodromal symptoms, we have already spoken incidentally. These may pass unnoticed for a time, but in a few weeks the patient's condition attracts attention. He is evidently not the man he was. His memory for recent events is much impaired; he consequently gets into confusion with his

domestic, social, and business arrangements, and his temper becomes unbearable. He sometimes appears to be conscious that his mental powers are not to be depended on, and he makes great efforts to maintain his part in general conversation, but these efforts only increase his embarrassment, and he may at last feel so mortified as to burst into tears. His condition is in many respects like that of a man slightly affected by drink, but who, conscious of his state, attempts unsuccessfully to conceal his mental confusion and muscular unsteadiness by acting as if nothing ailed him. Such a state, however, is rather the exception. The patient is usually much pleased with himself, and is very loquacious. Being forgetful, he repeats his stories until his hearers lose all patience, and he is then exceedingly irritated that his efforts are not sufficiently appreciated. A frequent and early feature is that he forgets the proprieties of life, and is indelicate and often indecent in his conversation and conduct.

Sooner or later, generally in the course of a few weeks, the second stage is reached. The patient has been passing further and further from a normal mental state; his general excitement and restlessness have been increasing; he is sleepless, and at last breaks out into acute mania. The mental symptoms are now as a rule characteristic. There is no limit to his ideas of his own power, riches, stature,

comeliness, &c. No subject can come into his head but it is magnificent. If any suggestion is made, it is at once adopted and improved. This is very characteristic. In ordinary acute mania it is common enough to find a man saying extravagant things, but the field is limited in each case. In a general paralytic, on the contrary, there is no end to the exalted delusions; they vary from hour to hour, from minute to minute, and are no sooner uttered than forgotten.

The degree of excitement varies much, from good-humoured loquacity to violence and restlessness which threatens danger to the patient and those around him. A man who believes himself to be the strongest person in the world, to have legs a mile long, and to be able to demolish the asylum in five minutes, is not generally slow in giving proofs of his powers. The management of such cases in this stage is accordingly difficult and dangerous; control is badly borne and may increase the excitement to the wildest fury. All the symptoms of acute mania, or more rarely of acute melancholia, are present. The patient is sleepless, dirty in his habits, destructive, and possibly refuses his food. He may appear thin, haggard, and pale; or on the contrary, in good condition, the face and head flushed, the eyes injected, the pulse full and quickened, and the temperature probably slightly increased.

This tempest of excitement may continue from one to three or four weeks and then gradually subside. The patient has now passed into the third stage, the longest of them all. His mental failure is evident enough, and it becomes progressively worse. The extravagant delusions are as marked as ever, though expressed with less vehemence. He often feels supremely happy, and his bodily health appears to him so perfect that mere existence is a constant source of pleasure. If his health be inquired after, it is "first-rate," "splendid," &c.

The physical symptoms, from which the disease receives its name, are now sufficiently marked. The ataxy is generally specially pronounced in the organs of speech. There is a thickness, an awkwardness in pronouncing certain words which need only be heard once to be readily recognised. The words are uttered with a deliberation which suggests slight intoxication. Special difficulty is experienced in enunciating such words as abound in consonants; thus, to settle the diagnosis it is usually sufficient to ask the patient to repeat "truly rural." He does not stammer, but he so blurs the r's that the words are almost unintelligible. The quivering of the muscles about the angles of the mouth is most distinctive. Even when the patient is silent, and all the features should be at rest, the angles of the mouth twitch

irregularly, and sometimes so violently that he appears to be about to weep. If asked to put out his tongue he may not be able to do so, or it is jerked out and back again rapidly. As it lies in the floor of the mouth, its surface is seen to be in a state of constant agitation, the result of the fibrillary movements of the subjacent lingual muscles. The handwriting is generally markedly changed, sometimes even at an early stage of the disease, and it becomes so irregular as to be illegible. In a patient suspected of being a general paralytic it is of great importance to examine the writing, for we may thus discover not only signs of ataxy of the muscles of the fingers, &c., but clear indications of mental failure. Words may be repeated or omitted so constantly as to prove at once that the mind is affected.

The gait becomes markedly ataxic. The feet may not always be lifted and put down as in a typical case of locomotor ataxy, but the muscular inco-ordination is evident enough in other ways. The patient cannot walk along a line, turn quickly round, or mount a chair. This disorder of locomotion may be very slight at first, but it gradually becomes worse, and at last the patient cannot walk at all, or even stand.

Now, though the general rule is for the disease to pass from bad to worse, in the more chronic cases surprising improvement not unfrequently occurs. All

mental symptoms may disappear, and all the more marked physical symptoms may do likewise, so that it would be easy for an inexperienced observer to believe that recovery had really taken place. There is, however, almost always some sign remaining to show that a remission only has occurred and not a true recovery. It is very seldom indeed that the inequality of the pupils entirely disappears. Still, these remissions are sometimes so complete and so prolonged, extending from a few months to two or three years, that sanguine writers have been tempted to record them as recoveries. A genuine recovery is unknown. If the diagnosis at first were correct, it is perfectly certain that the case will at last end fatally.

During the earlier part of this third stage the mental symptoms may vary considerably. Transient attacks of excitement of more or less severity frequently occur, and after each attack the mental and physical condition is found to have suffered. In marked contrast to the supremely happy state of mind usually observed, symptoms of melancholia or of hypochondriasis may appear, and the patient then requires constant attention to prevent suicide. In this depressed condition the delusions may be very extraordinary.

Though these attacks of excitement or depression may occur from time to time, the prevailing state is that of an ever-increasing dementia. The patient is

as happy as a king; though scarcely able to speak or walk, he says that he is "first-rate, and never felt better." He loses control over his sphincters; is so ataxic that he cannot button his clothes, and soon requires the attentions usually devoted to young children. He is at last confined to his chair, and, in the course of a few weeks or months, to his bed. His condition is now miserable indeed. In spite of all possible care bed-sores may develop rapidly. He can scarcely move himself, cannot feed himself; he emaciates, and his limbs draw up and become rigid. Fortunately the patient appears to be unconscious of his miserable plight. As long as his language can be understood, he is repeating to himself "happy, happy; millions of millions," or some such phrases as clearly indicate that as long as a trace of mind remains, he is rejoicing in his old dreams and delusions, and that he neither appreciates his lamentable mental state nor feels the physical infirmities which are rapidly ending his existence.

This third stage varies much in duration. Should the patient enjoy the benefit of careful nursing, it is sometimes wonderful how long his existence can be prolonged. In addition, should excitement, congestive or epileptic attacks, and other indications of acute changes in the nerve-centres, remain absent, this stage may be prolonged for years. On the other hand it is not unusual for the whole disease to run its course in a few months.

However degraded and demented the patient may become he is seldom so utterly lost as to lose his desire for food, and to manifest satisfaction at getting it. On the contrary, most general paralytics have ravenous appetites, and devour their food in great haste. Hence arises a great danger of choking. The mucous membrane of the throat has lost much of its sensibility, and thus the reflex movements of the pharynx and œsophagus are markedly diminished in rapidity and power. Hence if the patient be left to himself and supplied with ordinary food, he is almost certain to be choked, through the food being crammed into the mouth in such large pieces and so rapidly that it cannot be swallowed. Care should be taken that he gets only minced and pulp food; a dry morsel, such as a crust of bread, is specially dangerous.

During the continuance of the third stage several very characteristic features are developed. One of these is convulsions. Even in the same patient the convulsive attacks vary from time to time in their character. They may be examples of the *petit-mal*, or of the *grand-mal*; they may be general, unilateral, or confined to a single limb or group of muscles. Death may be directly due to the severity and number of the fits. The patient is suddenly seized by intense general convulsions; one fit may be scarcely over before another begins. As his strength fails, the fits are

probably reduced to constant muscular tremors of the whole body. Unconsciousness is profound; no food can be taken; there is great rise of temperature; the patient is bathed in a copious perspiration which often rises in a visible steam from his body; congestion of the lungs rapidly sets in, and he may die in two or three days from the beginning of the attack.

As a contrast to the condition above described, another patient may be slightly convulsed in his legs, he does not lose consciousness, he is able to sit on a bench, but cannot sit still. Every few seconds he starts as if an electric shock were passed through him. He is greatly puzzled at his state.

Another characteristic feature of this stage is the occurrence of transient attacks of cerebral congestion. The symptoms vary greatly in intensity, from slight drowsiness to profound coma. In the more serious attacks death appears imminent; yet in a wonderfully short time the patient rallies, and is able to walk about, although his mental and bodily condition are evidently worse, and his body may lean to one or either side as the result of paresis of some of the muscles of the trunk.

Even though a patient escapes all the complications referred to, his end comes at last. In his feeble state climatic changes are badly borne;

a slight fall in the temperature may cause a pulmonary congestion, which is rapidly fatal; or he may simply die from asthenia.

TREATMENT.

All that can be done in the present state of our knowledge is to treat symptoms as they arise. In some cases the course of the disease is so quiet that careful nursing is all that is required. But in many others the excitement and other distressing symptoms necessitate removal to an asylum. When the excitement is slight, rest in bed is often quite sufficient, but seclusion may be required when it is extreme, or when his violence is dangerous to the patient or those around him. Drugs may also be used at the same time. Some physicians place great reliance in *Tr. Digitalis*, giving it in m. xv—m. xxx doses every three or four hours. Another most useful remedy is *hyoscyamine*. It must be given with great caution, and the first dose should not exceed $\frac{1}{4}$ gr. According to the effect produced the dose must be increased or repeated. In other respects the treatment during the stage of delirium does not differ from that pursued in other diseases. The strength must be maintained by nourishing food; and if the patient refuse it, he must be fed by the stomach-pump.

As the convulsive seizures often directly threaten

life it is important to know that they can almost always be arrested by the administration of chloral. Cold to the head and spine is often useful, but chloral is *the* remedy. Thirty grains in water will almost always bring the attack to an end. If the drug cannot be swallowed, it should be administered per rectum, and repeated if necessary

CHAPTER XXI.

EPILEPSY.

THE convulsive affection to which reference is made in this chapter is usually considered to be of functional origin, and often gets the name of idiopathic epilepsy in contradistinction to the fits or convulsions which occur as hysterical manifestations on the one hand, and as the result of a determinable organic disease of the brain on the other. The pathology of idiopathic epilepsy is exceedingly obscure. Indeed, the disease may be said to possess no morbid anatomy, for the histological changes which have been observed from time to time are not only not constant, but must be regarded as secondary to the disturbance of function rather than its cause. Thus the dilatation of the capillaries of the medulla, described by Schroeder van der Kolk, and the pigmentation of the cells of the same region, observed by Voisin, have very little significance; and the same may be said of the sclerotic changes found by Meynert in the *pes hippocampi*. Certain writers have endeavoured to furnish the disease with an anatomical substratum to be found in the cerebral cortex; nevertheless, although experimental

research might favour such a view, its supporters must content themselves with the theory that the lesion, should such exist, is a molecular one, for no constant histological changes have been met with here either. The current theory of epilepsy appears to be that all the phenomena are caused by a sudden discharge of nerve force, or liberation of energy, from certain infirm motor cells, situate in the gray matter, most frequently in the cortex, though possibly in some instances lower down, and even in the medulla. According to Gowers, whose classical treatise on epilepsy deserves the highest possible commendation, the vaso-motor theory, or the supposition that the loss of consciousness and convulsions are due to vaso-motor spasm, is untenable. Todd was the first to attribute the seizures to an explosion of nerve force, and this theory was afterwards elaborated by Dr. Hughlings-Jackson, who advanced the view that convulsions of all kinds attended by loss of consciousness were caused by discharging lesions of the cortex of the brain.

ETIOLOGY.

In considering the causes of epilepsy it is most important to recollect that the occurrence of a fit renders the patient more or less liable to a return in future; just as a dislocation of the head of the humerus, to borrow an illustration from

surgery, paves the way for, or makes easier the occurrence of, a future dislocation, so the first fit lays the foundation for the second, and so forth. From this point of view the etiology of the disease in any case practically resolves itself into the causes of the first seizure. Hereditary predisposition can very often be traced. There may not be a history of epilepsy, but it is very common to meet with some allied neurotic tendency in the family, such as insanity, hysteria, chorea, &c. In the great majority of cases the fits commence in early life. Dr. Gowers, who analysed 1,450 cases,* found that 29 per cent. began under ten years; about 46 per cent. between ten and twenty; and in the next decennial period, from twenty to thirty, only 15·7 per cent. Females suffer from epilepsy rather more frequently than males, and are more exposed to hereditary influences (Gowers). Scrofula and rickets undoubtedly predispose to the occurrence of epilepsy, and, according to Sir William Jenner and others, the unstable nervous system which accompanies the latter condition is responsible for most of the convulsive attacks usually ascribed to dentition. My own opinion is decidedly in favour of this view; and from my experience at the Newcastle Children's Hospital I have been led to believe that the defective nutrition associated with rickets exercises

* Gowers : "Epilepsy," p. 13.

an enormous influence upon the production of epilepsy. Fright, excitement, and anxiety are very common *exciting* causes, and they necessarily operate most frequently in individuals where a strong predisposition to the disease exists. The same may be said of the other causes to which the occurrence of idiopathic epilepsy has been assigned, such as masturbation, chronic alcoholism, intestinal irritation (worms, &c.), traumatic influences, acute diseases, &c.

SYMPTOMS.

True epilepsy is characterised by attacks of temporary loss of consciousness of sudden onset, along with which are usually associated tonic or clonic spasms.*

Two kinds of seizures are described, the severe or major attacks, or *grand-mal*; and the minor attacks, or *petit-mal*. Premonitory symptoms by which the patient is warned of the approaching fit occur very frequently, and it will be convenient to refer to them before describing the paroxysmal attack. These symptoms may show themselves a day or two before the seizure, or may precede it by a few seconds only. The warnings which occur immediately before the epileptic attack are termed *auræ*. The more remote warnings consist in an

* This definition will not hold in every instance, as in some of the minor attacks consciousness is not completely lost.

altered mental state, such as irritability of temper, or the reverse; cold feet; headache and giddiness; loss of appetite, &c.

The auræ most frequently noticed are thus classified by Gowers, who states that these warnings occur in about half the cases:—“(1) The unilateral auræ, a motion or sensation in one side of the tongue, face, trunk, or in one arm or one leg. (2) Certain general auræ; bilateral sensations in the limbs, tremors, starts, malaise, faintness, &c. (3) Auræ referred to certain organs, mainly to those to which the pneumogastric nerve is distributed, and to this group belong most of the visceral warnings. The most common is the well-known epigastric sensation, and others are a feeling of choking, dyspnœa, nausea, and cardiac sensations. (4) Vertigo and other allied sensations. (5) Certain sensations in the head, pain, &c. (6) Psychical auræ, the consciousness of an emotion or an idea. (7) Special sense warnings.” Space is too limited to allow of a more extended notice of the auræ, therefore I must refer the reader to the chapter on the subject in Dr. Gowers’ book, which will amply repay a perusal.

The minor paroxysms, or attacks of *petit-mal* are, as a rule, only momentary, and consist in sudden loss of consciousness without marked spasm. According to Brown-Séquard there is always some

slight motor phenomenon, but Russell Reynolds and others assert that the attack may consist alone in momentary "loss of perception and volition." The seizures are so slight that the patients commonly designate them as "sensations," "turns," "nerves," "faints," &c. In such an attack the person may or may not fall, very often the erect posture is maintained, and, indeed, he may continue to support any object held in the hand. In some cases pallor of the face is observed. Auræ may precede these minor attacks as well as the severe ones, giddiness being the most common. The return to consciousness may be sudden, and the patient may continue at once whatever he was occupied with before, so that the fit may pass absolutely unobserved by the bystanders. But very often a confused state of mind is left from which the patient recovers slowly, or he may fall asleep. In other cases, again, post-epileptic phenomena are manifested, such as the automatic performance of certain actions, or the occurrence of hysteroid convulsions. This *automatism* is of practical importance on account of its medico-legal aspect, as the actions may be complex and have a voluntary aspect (Gowers). Articles have often been stolen in this state, and other seemingly criminal acts performed. One of my patients invariably changed his clothes; indeed, undressing is a very common post-epileptic action. Trousseau

relates the case of an architect who frequently had an attack when on a lofty scaffolding; when seized he would run quickly along the planks shouting his name, but he never fell.* I have seen an epileptic suddenly rise up in a crowded assembly, then jump upon the back of a bench and run from bench to bench for several yards.

The hysteroid attacks (the name given by Roberts to epileptic paroxysms of an hysterical type) which follow epileptic seizures are generally wild "hysterical" spasms in which the patient throws his arms and legs about in a co-ordinated manner.

In many cases, as already indicated, an aura precedes attacks of *grand-mal*, but in some consciousness is lost so suddenly as to prevent the recognition of any warning. The patient falls down as if struck by lightning, often uttering a cry as he falls. At once the muscles of the eyes, face, neck, chest, and limbs become affected with tonic spasm. The head and eyes (which are wide open) are turned to one side, the limbs become rigid and fixed, and the breathing stops, while the face, previously of a deathly pallor, becomes cyanotic. The pupils are dilated and the pulse is small. This stage lasts, as a rule, from ten to thirty seconds, when it is succeeded by a period marked by clonic spasms, the transition to which is abrupt. The first muscles to be affected by this intermitting

* Trousseau : Clinical Lectures, translated by Bazue, vol. i., p. 59.

spasm are usually those of the face, tongue, larynx, pharynx, and chest. The irregular jerking spasms that ensue make the patient hideous to observe, as every feature is horribly distorted. The jaws are violently champed, the tongue frequently bitten, and the saliva exudes from the mouth in the form of bloody foam. The facial lividity is now intense, and death appears imminent. Gradually the intervals between the spasmodic shocks lengthen, and air begins to enter the thorax. Soon the spasms cease, and the third stage is entered upon, that of utter prostration, during which the patient usually sleeps heavily for a time, and then begins to show signs of returning consciousness. The convulsive part of the seizure frequently lasts four or five minutes, but in some cases longer. Some patients sleep for many hours after a fit, while others recover consciousness almost immediately. Urine is often passed during the paroxysm.

There is a great variety in the frequency and severity of the fits in different cases; and the degree to which the mental and physical vigour is impaired also differs considerably. In some cases the attack only recurs every few months, in others the interval is much shorter. In some there is a tolerably marked periodicity: in a patient at present under my care, for example, the fits have occurred on Thursday night, once a fortnight, for

nearly fourteen months. It is very common to meet with cases in which attacks of *petit-mal* occur from time to time during the intervals between the severe paroxysms. The fits very often occur at night, and it must be recollected that some patients are the subjects of such seizures without being aware of it. Gowers records the case of a patient who suffered from convulsive attacks at night every few months for eighteen years, and yet he was in complete ignorance of the fact. The general health of epileptics is rarely very good between the attacks, they often complain of dyspeptic symptoms with more or less giddiness and headache. As the disease progresses the distinct psychical disturbances are manifested, which may advance to complete dementia. Whilst there is generally some mental impairment, it is certainly a fact that in the case of some patients this is by no means obvious.*

DIAGNOSIS.

Idiopathic epilepsy has to be distinguished from the following conditions:—Attacks of syncope; hysteroid seizures; lead poisoning; uræmia; convulsive attacks arising from gross cerebral disease, such as tumour; reflex convulsions; general paralysis of the insane; and simulated or feigned fits.

Minor seizures may readily be mistaken for

* Julius Cæsar and Napoleon both suffered from epilepsy.

syncopal attacks, in fact, many patients designate them "faints." Attention to the fact that the period for which consciousness is lost is much shorter in epileptic than in fainting attacks, and also that the former are often preceded by some distinctive aura, will generally enable a diagnosis to be made. The pulse does not become so feeble in *petit-mal* as in syncope.

Some hysterical seizures resemble true epilepsy very closely; others, again, are easily recognised. To the former, the border-line cases, the term "hysteroid" seems specially applicable; moreover, many cases of this sort are genuine epilepsy with hysterical tendencies. The following features are characteristic of the purely hysterical paroxysms:—consciousness is perverted or disordered rather than lost completely; unilateral auræ are very rare,—when any warning is observed it is usually palpitation, choking, or bilateral foot aura; the onset is often gradual, the convulsions are wild and purposive (co-ordinated), the spasms suggesting a struggle rather than a true fit, and often tonic and clonic spasms alternate; the patient may talk and bite, but she rarely bites her own tongue; micturition never happens; the attack may last a long time, and can often be controlled. It must be borne in mind that many true epileptic attacks are followed by post-epileptic hysterical phenomena. Plumbic and uræmic convulsions can generally be dis-

tinguished without difficulty from epilepsy, as the discovery of the signs of these toxæmic conditions will suggest a diagnosis, and further, in lead-impregnation and uræmia the attacks often last for hours or days, during which time the fits follow one another at short intervals, constituting a toxæmic "*status epilepticus*."*

It is very important to discriminate between organic fits and idiopathic epilepsy. The convulsions of organic disease are often unilateral; the aura is very frequently unilateral, and often visual or auditory (Gowers). In many cases consciousness is not lost; and post-epileptic paralysis often lasts for a considerable time after the seizure, in other words the convulsed limbs remain more or less helpless for a time. There will generally be a history of headache and vomiting between the fits, and optic neuritis and other signs of a gross lesion will usually be found. The diagnosis of true *reflex* convulsions is often very difficult. The causes which operate in this direction are dentition, intestinal worms, injudicious food, uterine displacements, &c. The purely reflex epileptiform seizures are sometimes succeeded by lasting epileptic convulsions. Feigned fits are not difficult to detect, but care should be taken lest the apparent imposition be really an erratic form of true epilepsy.

* Occasionally, in both lead and kidney cases, the convulsions resemble ordinary epilepsy very closely in the way in which the fits occur.

PROGNOSIS.

The danger to life during a paroxysm is comparatively slight, but still a certain risk exists, for the patient may roll on to his face after a fit and be suffocated, or he may choke, should an attack occur during a meal; and finally, there is always the danger of falling from a height, or into water or fire.

The fact is apparent to all who have had much to do with the treatment of the disease, that the probability of a cure is very slender indeed; or in other words, the prognosis of epilepsy is extremely unfavourable. It must be allowed, however, that occasionally a patient does recover, and according to Gowers the following are the lines on which a prognosis must be made:—Slightly more males recover than females; the cases which commence after twenty years of age are rather more hopeful than those beginning before that period, though the prognosis is a little better in those which commence under ten than between ten and twenty; “the prognosis is favourable in the inverse proportion to the duration of the disease,” the longer the affection has lasted the more remote are the chances of recovery; cases in which hereditary predisposition exists are rather more favourable than others, though it must be stated that Brown-Séguard does not seem to favour this view;

the more frequent the fits the worse is the prognosis; there is more hope if the attacks occur in the sleeping or waking state only than if they occur in both; the existence of an aura seems to be favourable; and finally, the prognosis is rather more propitious if the mental state be good, and if the attacks are all of the same variety. Epilepsy commencing in childhood from teething or a bowel-complaint, and having lasted many years, is almost incurable (Brown-Séguard).

TREATMENT.

During a fit the patient should not be interfered with, it being alone necessary to loosen the neck-clothes, and to keep him from biting his tongue or otherwise injuring himself. Some attacks can be aborted or warded off when they are preceded by a definite aura or warning. Thus, if a sensory aura is felt in a limb the tight application of a band above the part in which it has started may prevent the seizure. In some cases it is more successful to tie and untie the ligature very rapidly, repeating the process several times, than to leave it fastened on the limb. Brown-Séguard has shown that pinching or otherwise irritating the skin answers equally well. When the aura is a motor phenomenon—the contraction of a group of muscles—the fit may be arrested by forcibly extending the contracted limb. For example, there is a patient

at present in the Newcastle Infirmary who sits for hours at a time alternately extending and flexing the fingers of his right hand, with his left, in order to ward off a threatened right-sided spasm, which was wont to commence in the hand.

In treating a case of epilepsy we must in the first instance endeavour to ascertain and remove the morbid conditions upon which the fits depend, and then attack the seizures themselves in a more direct manner. After the removal of any local or general morbid condition which might possibly occasion epileptic fits, the administration of one or more of the following remedies may be of service in arresting or mitigating the attacks. The bromides deserve the first place, and the potassium salt is held in most esteem, though it does not contain so much bromine as the ammonium and lithium preparations.* I have used the latter extensively, and I do not think it possesses any advantage over the potassium salt; indeed, I quite agree with Gowers in the opinion that the relative power of the bromides in arresting epileptic fits is not proportional to the quantity of bromine which they contain. It is customary in ordering bromide of potassium to prescribe three or four times a day the minimum dose which will arrest the fits—usually from 20 to 40 grains to the dose for adults,

* Bromide of lithium contains 92 per cent. of bromine; bromide of ammonium, 81 per cent.; and the potassium salt only 67 per cent.

and less in proportion for children; but as Niemeyer pointed out, taking his *cue* from a celebrated quack, it is most essential to increase the dose from time to time. Of late Gowers has adopted what he terms the “method of *maximum dose treatment*” with a considerable degree of success. This plan consists in the administration of large doses of bromide (two or three drachms) every third or fourth morning, and by degrees the quantity is increased up to six drachms or an ounce every fifth morning, the maximum dose being reached in two or three weeks, and repeated several times, and then gradually reduced, the whole course lasting six or seven weeks. The large doses must be well diluted.

Brown-Séguard highly approves of the simultaneous employment of tonic remedies, such as strychnine, arsenic, &c., with the bromides. The following is a favourite prescription of his:—

R. Potassii iodidi ʒ ij
 Potassii bromidi ʒ i
 Ammonii bromidi ʒ iij
 Potassæ bicarbonatis ʒ i
 Tinct. Calumbæ ʒ i
 Aquæ destillatæ ʒ vi.

Of this he gives four doses a day, three one table-spoonful doses, and a large dose of three table-spoonfuls at bedtime. The same authority emphasises the statement that the bromide treatment must be persevered with for at least two years

after the appearance of the last attack. In some cases the bromide of zinc deserves a trial in gradually increasing doses up to twenty grains three times a day. In whatever form the bromide is used it is most essential that the treatment should be continued for a long time, care being taken to avoid *bromism*. *Arsenic*, *digitalis*, and *belladonna* are all of service in combination with bromide, but seldom of much value alone; the first mentioned will often prevent the bromide rash. The *zinc* preparations, especially the oxide in doses of three to six grains twice or thrice daily, sometimes do good. Iron, by improving the general condition of the patient, is frequently beneficial.

CHAPTER XXII.

LABIO-GLOSSO-LARYNGEAL PARALYSIS.

THIS affection consists of paralysis and wasting of the muscles of the tongue, lips, larynx, and pharynx, the result of a lesion in the medulla of the nuclei of the hypoglossal, facial, spinal accessory, and glosso-pharyngeal nerves. Two forms have been described, the acute and the chronic. The latter may appropriately be considered first, as ordinary *bulbar paralysis*, in the usual acceptance of the term, is a chronic malady.

CHRONIC LABIO-GLOSSO-LARYNGEAL PARALYSIS

(*Progressive Bulbar Paralysis*).

MORBID ANATOMY.

The ganglionic cells of the nuclei of origin in the medulla of the nerves of the affected muscles become shrunken and wasted; their nuclei and processes disappear, and the whole nucleus is more or less degenerated; whilst the nerve fibres themselves undergo atrophic changes. The degeneration is most pronounced in the hypoglossal nucleus, whence we have generally the starting-point of the

lesion, which involves next the spinal accessory nucleus, then the ganglion cells which are related to the lower filaments of the seventh,* and subsequently the nucleus of the glosso-pharyngeal.

The fibres of the affected muscles are very pale, and may exhibit granular degeneration; whilst the muscles generally diminish in bulk, unless the interstitial tissue be markedly increased, which seldom occurs. Of course it must be borne in mind that the lesion in the bulb is often associated with progressive muscular atrophy and other cord affections.

ETIOLOGY.

The causes are obscure. The majority of the cases occur in men over forty; the disease is very seldom met with under that age, and somewhat rarely in women. Syphilis is said to play a leading rôle in relation to the affection, but the following may be mentioned as amongst the most important of the immediate causes:—injury to the back of the neck, exposure to cold, protracted use of the muscles of the lips, tongue, and larynx, as in singing, mental anxiety and excitement, &c.

* Named the accessory nucleus of the seventh by Ross.

SYMPTOMS.

Occasionally headache and pains in the neck, with a feeling of constriction in the throat and chest, especially when speaking, initiate the more characteristic symptoms of the disease. By degrees a difficulty is experienced in speaking and eating. At first there is but slight indistinctness of speech, due to imperfect pronunciation of the dental-palatine consonants—d, t, l, n, s. As the difficulty increases, the labial explosives, b, p, v, are imperfectly formed on account of weakness of the lips, the vowels o and u cannot be distinctly sounded, and prolonged speaking is attended by marked fatigue, whilst whistling becomes an impossibility. As a further development of the affection the voice becomes nasal, and liquids are regurgitated through the nostrils, the result of paralysis of the velum palati; and at a still more advanced stage, when the constrictors of the pharynx become involved, difficulty in swallowing is complained of, and the dysphagia is greatly exaggerated when the muscles which elevate the larynx and close the glottis are paralysed. By degrees the voice becomes more and more feeble and indistinct, until at length it is reduced to unintelligible sounds. As the saliva is secreted more actively than in health (Rosenthal), and cannot be swallowed, it dribbles over the

lower lip, to which the patient usually has his handkerchief applied.

In addition to being paralysed, the tongue and lips generally suffer emaciation, and fibrillary movements are common. In some cases, however, there is no diminution in bulk; but the observer must not here be deceived, for, whether atrophy be apparent or not, there is always wasting of the muscular tissue, though this may be compensated for by an increased development of fat. As a rule, the sensibility of the affected parts remains normal, though in some rare cases the nuclei of the *fifth* are implicated, when trigeminal anæsthesia results. Usually the electrical reaction of the paralysed muscles is unaltered, though Erb has found the reaction of degeneration. Of necessity when extreme atrophy is present a quantitative alteration must be expected. As the disease advances general emaciation ensues from impaired nutrition and insufficient food, and death may result from paralysis of the respiratory muscles, inanition, or choking. Occasionally paralysis of the heart terminates the case. As in the case of ordinary progressive muscular atrophy (*see* p. 326), the lesion may extend to the medulla and occasion bulbar symptoms; so it is not uncommon for the former affection to supervene upon labio-glosso-laryngeal paralysis.

DIAGNOSIS AND PROGNOSIS.

Bulbar Paralysis is difficult to recognise with certainty at an early stage, but when the disease is fully established, the defective speech, the difficulty with which the tongue is protruded, the paralysed lips, the salivary flow, and the dysphagia leave no room for doubt.

The affection commences insidiously, and runs a slow and almost certain course towards a fatal termination, which supervenes in from one to four years.

TREATMENT.

No case of genuine progressive bulbar paralysis has been known to recover, so our efforts can only be directed against the lesion with the view of retarding its advance. Foremost amongst the remedies which may be tried for such a purpose Erb recommends the employment of the continuous current, as follows:—the current to be passed transversely through the mastoid processes, and longitudinally through the neck, the positive pole to be applied to the upper part of the neck, and the negative to the pharynx externally, *i.e.*, beside the angle of the jaw, so as to induce swallowing movements. In addition, galvanic and faradic currents should be applied direct to the paralysed muscles. The ordinary nervine tonics may be

administered internally. Rosenthal and others favour cold douches to the neck while the patient is in a warm bath. Only soft, nutritious food should be given to the patient, and great care exercised to prevent him from choking; indeed, towards the termination of the case it may be necessary to feed him through a tube.

ACUTE LABIO-GLOSSO-LARYNGEAL PARALYSIS

(Acute Bulbar Paralysis).

This is an acute lesion that gives rise to paralysis of similar distribution to that of the chronic variety. Few uncomplicated cases have hitherto been recorded, and though it is difficult to remark with certainty upon the precise character of the lesion, it is nevertheless probable that inflammatory processes, embolism, or hæmorrhage underlie the majority of the cases. In Leyden's cases small centres of softening were found. To explain the fact that a very limited lesion may give rise to paralysis of the tongue, lips, pharynx, and larynx, Gowers suggests that the ascending fibres from the nuclei which supply these parts may pass up close to the middle line and thus be exposed to damage as a group.

SYMPTOMS.

In this disease the patient experiences a sudden difficulty in speaking and swallowing, and at the same time his tongue becomes paralysed so that he cannot protrude it. The attack though sudden is rarely apoplectic, in the sense that consciousness is lost. Sudden bulbar seizures of this description are very often fatal within a few hours or days; however, death may occur almost immediately, the patient falling down with a cry, or in convulsions. In the slighter cases, viz., those in which the onset is survived, there may be weakness in the limbs along with paralysis of the tongue, lips, and throat. But the former often passes off, leaving labio-glosso-laryngeal paralysis, as in the case of the chronic affection; only there is less wasting, and the paralysis is not progressive.

DIAGNOSIS, PROGNOSIS, AND TREATMENT.

The sudden seizure, accompanied by symmetrical paralysis of the tongue, &c., cannot readily be mistaken.

The prognosis is necessarily bad even in the slighter attacks, though it should be recollected that the same tendency to advance is not manifested as in the chronic form. The syphilitic cases are certainly the most hopeful.

Very little can be done at first in the shape of treatment beyond absolute rest and the application of cold to the back of the head and neck. At a later period the muscles should be treated electrically.

PART II.

THE SPINAL CORD.

CHAPTER XXIII.

ANATOMICAL AND PHYSIOLOGICAL
INTRODUCTION.

THE relation of the spinal cord to the bones which form the canal in which it lies must receive our careful attention. The cord may be said to commence at the top of the vertebral column, and to end between the first and second lumbar vertebræ. The cervical enlargement of the cord commences about an inch below the foramen magnum, and ends opposite the seventh cervical vertebra. The lumbar enlargement corresponds to the eleventh and twelfth dorsal and first lumbar vertebræ. The nerves do not leave the bony canal at the same level as their point of origin from the marrow, but, on the contrary, descend in the canal before issuing from it. The difference between the level of origin from the chord and of exit from the vertebral column increases as the lumbar region is approached.

Fig. 26 represents a section of the lumbar enlargement of the cord. The white columns consist of two anterior, two lateral, and two posterior,

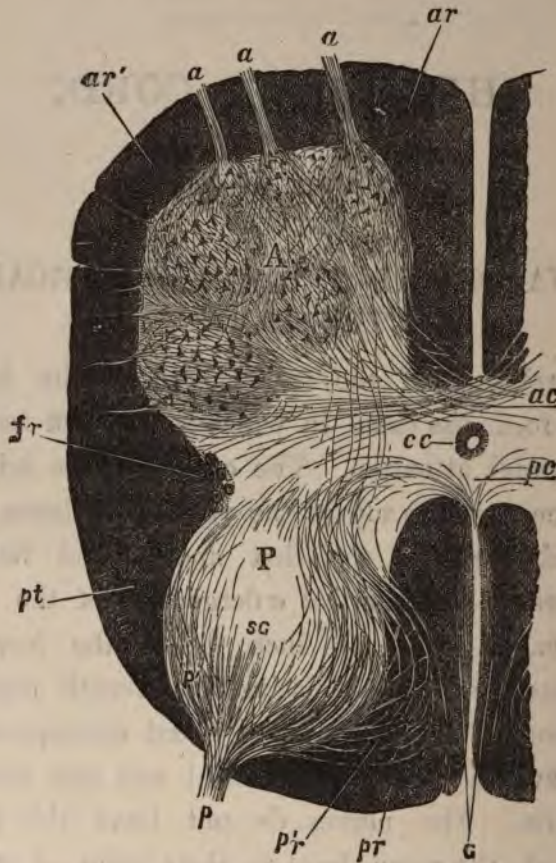


FIG. 26 (Ross).—Section of Spinal Cord from the middle of the Lumbar Enlargement.—A, P, anterior and posterior gray cornua respectively; SG, substantia gelatinosa; cc, central canal; ac, pc, anterior and posterior commissure respectively; G, columns of Goll; pr, posterior root-zone; p, posterior root; p', external radicular fasciculus; p'r, internal radicular fasciculus; a, a, a, anterior roots; ar, ar', anterior root-zone; fr, formatio reticularis; pt, pyramidal tract; T, column of Türck (fig. 27).

which last in the upper dorsal and cervical regions are subdivided into the columns of Goll, or posterior median columns, and postero-external columns or root-zones. The gray matter has anterior and posterior horns, from the former of which the anterior or motor nerve-roots pass out in an irregular fashion through the anterior white column that separates the horn or cornu from the surface. The posterior or sensory roots enter the cord in a more consolidated form, and make their way, some into the posterior horn, and some into the postero-external columns.

The motor fibres coming from the motor centres of one side of the brain divide into two parts at the medulla. One set (the larger) decussate, and pass down the lateral column of the opposite side until they reach the segment of the cord corresponding to the pair of nerves of whose anterior roots they are to form part, when they enter the gray matter, and pass out on the same side (fig. 27, Bramwell). The other set, about one-tenth or one-eleventh of the whole, continue down the cord in the anterior column on the side to which the fibres originally belonged, and either enter the anterior horn of the same side and pass out amongst the anterior roots, or else decussate in the anterior white commissure, and pass out with the opposite anterior roots. Authorities are not agreed respecting the

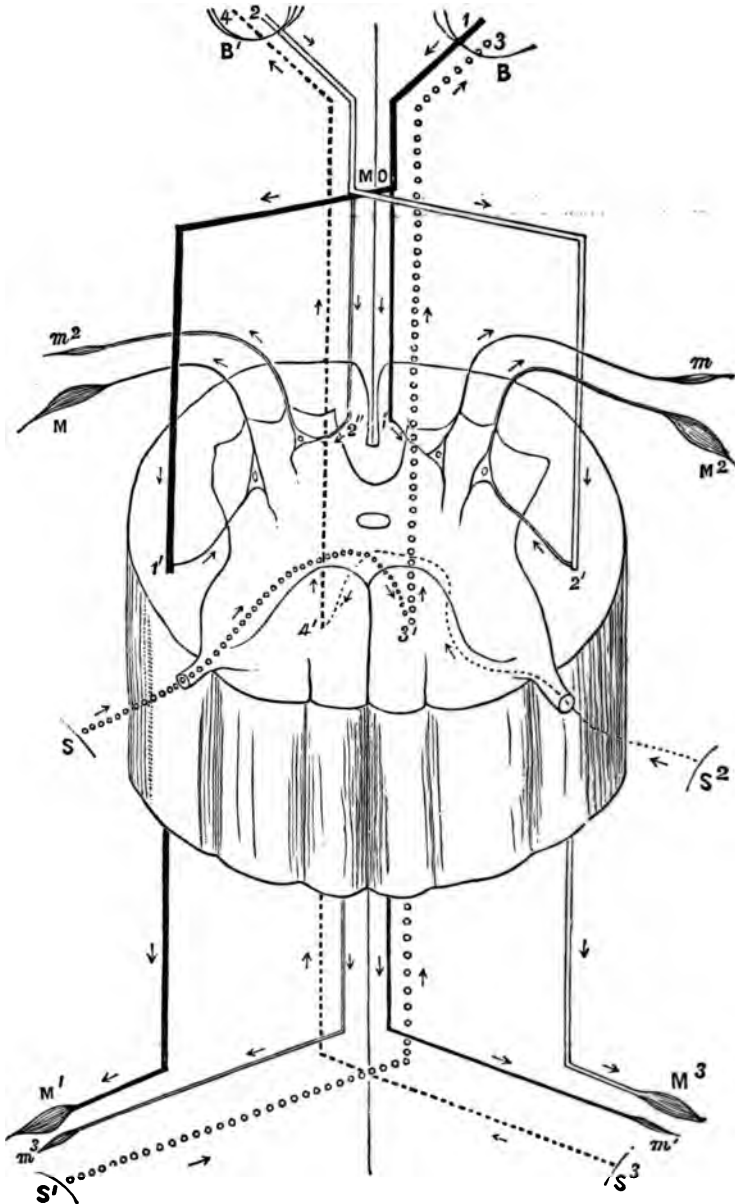


FIG. 27 (after BRAMWELL).—*Diagrammatic representation of the Spinal Cord as a centre and conducting medium.*—B, right, B' left, hemispheres of the brain.

1. Motor tract from the right hemisphere, dividing at MO; the larger subdivision, decussating, pass by means of the lateral column to supply the muscles M, and M', on the opposite side; the smaller bundle remaining on the same side pass down the anterior column to supply the muscles m, and m'.

2. Motor tract from left hemisphere, supplying the muscles M² and M³ on the right side of the body, and m² and m³ on the left. S, S', sensitive areas on the left side, and S², S³, sensitive areas on the right. The arrows indicate the directions in which the nerves conduct.

course of these latter, which form the so-called direct pyramidal tracts, or columns of Türck, T, fig. 28; and for the present we must reserve

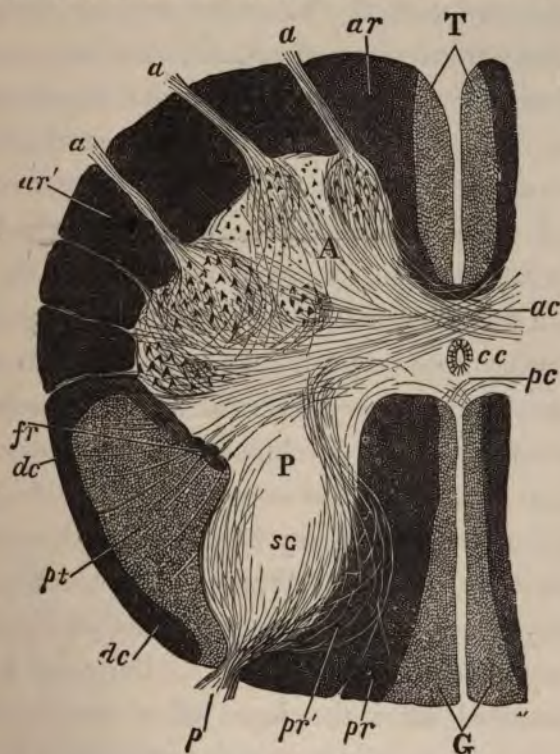


FIG. 28 (ROSS).—Middle of Cervical Enlargement.
The letters indicate the same as the corresponding ones in fig. 26.
T, columns of Türck; dc, dc, direct cerebellar tracts.

judgment upon the point in dispute. The posterior or sensory fibres split up into two bundles as they pass into the cord by the posterior root. One set make their way directly into the posterior gray horn, and then decussate in the gray matter to the opposite side where they ascend, principally in the posterior column (more especially, as some think, in the posterior median column, or column of Goll), but some ascend in the lateral column, and others in the central gray matter. The other set force their way into the postero-external column, and then enter the gray matter and decussate. Some fibres can be traced forwards from the posterior roots to the anterior cornual cells with which they are connected through the gray network of Gerlach.

We have thus an anatomical division of the cord into an antero-lateral or motor area, and a posterior or sensory area; but a further subdivision of the columns is necessary—a subdivision indicated by observations on the development of the cord, and by certain pathological processes, viz., the degeneration of motor and sensory fibres in the direction in which they conduct. When the motor fibres are destroyed, say in the crus cerebri, either by disease or experiment, certain tracts in the cord become degenerated after the lapse of a certain period. The degenerated fibres, which presumably form the principal motor tracts of the cord, are found in the anterior and lateral white columns; in the latter

they occupy the posterior portion of the column of the side opposite to the hemisphere from which they have travelled, and form the crossed pyramidal tract, composed of the fibres which, coming from the injured crus, have decussated at the anterior pyramids of the medulla. The degenerated fibres of the anterior column occupy the internal aspect of the column of the same side as the crus from which they have come. This tract is known as the direct pyramidal tract (or column of Türck)—*i.e.*, the fibres which have not decussated in the medulla. The white fibres of the lateral columns which constitute the outer margin of their middle portions are in communication with the cerebellum, and get the name of the direct cerebellar tracts (*dc, dc*, fig. 28). The division of the posterior columns into the postero-internal or columns of Goll, and the postero-external or root-zones, is also strictly physiological. The sensory fibres which degenerate in the centripetal direction as the result of a lesion of the sensory tracts, say in the upper dorsal region, occupy the internal or median aspect of the posterior columns—*i.e.*, the columns of Goll. These columns are supposed to be the principal tracts of sensory conduction. The postero-external columns are made up in a great measure of fibres which pass from the posterior roots to the gray matter.

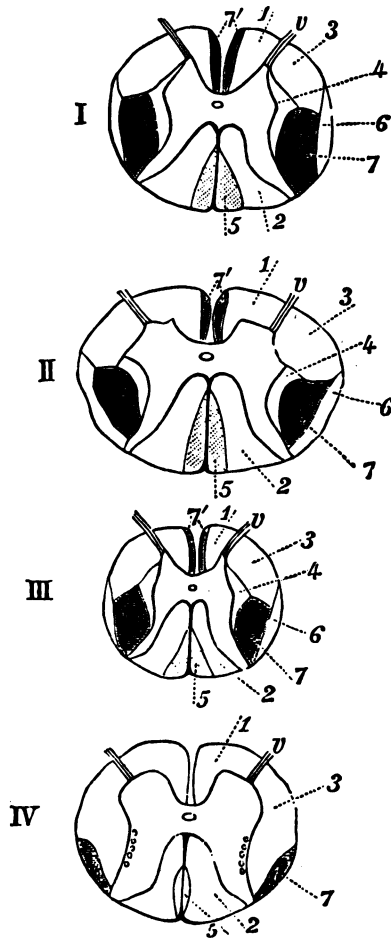


FIG. 29.—Diagram showing the different physiological tracts at various levels of the cord; and the order in which they are developed (after FLECHSIG).—I. Section at the height of the 3rd cervical; II. At the height of the 5th cervical; III. At the height of the 6th dorsal; IV. At the height of the 4th lumbar nerves.

1. Principal mass of the anterior column; 2. Wedge-shaped tract; 3. Anterior mixed region of the lateral column; 4. Lateral boundary layer of the gray substance; 5. Column of Goll; 6. Direct cerebellar tract; 7. Pyramidal tract of the lateral column; 7'. Pyramidal tract of the anterior column; v. Anterior root.

A few points respecting the functions of the different portions of the cord have yet to be referred to.

The large cells in the anterior half of the gray matter are extremely important, and will call for especial notice. They have several poles or processes:—the axis-cylinder process, which is really the origin of an anterior root-fibre (motor), and the protoplasmic processes, which form, by communicating with one another and with the posterior fibres,

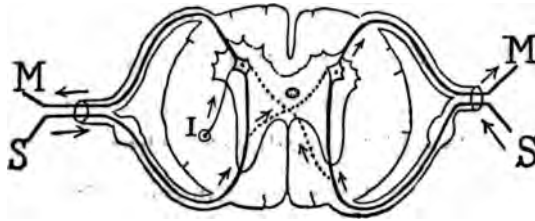


FIG. 30.

the dense network of gray fibres of which the gray matter is largely composed. Without being able to distinguish these cells anatomically, save by the gross arrangement of them into groups, it is tolerably certain that, physiologically speaking, there are different descriptions of large cells in the anterior cornua which serve as different centres. Thus we speak of motor, reflex, trophic, and vaso-motor centres. Fig. 30 is intended to represent a reflex arc which is made up of an afferent or sensory nerve, a nerve centre, and an efferent or motor nerve: M represents the

muscles, and S the sensitive surface. The dotted line is inserted to show how a crossed reflex act may occur.* The reflexes are generally described as consisting of two kinds, the so-called common, skin, or superficial, and the tendinous or deep.† The former are represented by the plantar, cremasteric, abdominal, epigastric, and other reflexes; and the latter by the patellar tendon-phenomenon, ankle-clonus, and other tendinous jerks. Some writers have urged that the afferent nerves through which the latter phenomena are carried on pass into the gray matter in the bundles of fibres which pierce the root-zones, whilst the afferent nerves on which the superficial reflexes depend enter by the bundle which pass direct to the posterior horn. The well-known reflexes of locomotor ataxy, viewed in the light of the morbid anatomy of that affection, are principally responsible for this view.

Reflex action may be inhibited by an effort of the will, when it is generally supposed that the fibres along which the inhibiting impulses travel are in the lateral columns (fig. 30, I); or it may, as a pathological phenomenon, be abolished entirely,

* A crossed reflex act ensues when the afferent stimulus crosses to the centre in the opposite side of the cord, and is transmitted by the motor nerves of that side; as when the tickling of the plantar surface of the right foot is responded to by a movement of the left limb.

† I have retained the term "deep reflex" for convenience; but I wish it to be understood that I do not believe the "knee-jerk" or "ankle-clonus" to be true reflexes. At the same time I must admit that the condition of muscle which is favourable or adverse to the production of the phenomenon depends upon spinal influences.

diminished, or increased. Any lesion which breaks the reflex arc connection will of necessity interfere with the reflex act, such as a peripheral one affecting either the afferent or the efferent nerve, or a central affecting the conducting part of the arc in the cord or the nerve cell itself. Reflex action may be exalted by an over-sensitive condition of the nerves or of the cell of the arc, or by the cutting off of the cerebral inhibiting influence. The trophic cells of the anterior cornua exercise a most marked influence upon the nutrition of the muscles and motor nerves; a lesion of these cells (as in infantile paralysis) is rapidly followed by marked muscular atrophy. Other portions of the gray matter (posterior horns) exert an influence upon the nutrition of the skin, &c.

The lumbar enlargement deserves a special notice. Fig. 31 represents diagrammatically the different centres located in this region of the cord, and their connection with the brain on the one hand and the organs over whose functions they preside on the other. The diagram is not intended to be topographically correct. The bladder centre has been supposed by Gowers to be composed of two sub-centres,—one for the sphincter, and the other for the detrusor muscle. A sensory impression ascending along the afferent nerve from the mucous membrane of the bladder will ascend to the brain, when an impulse descending in the motor tracts to

the centres in the lumbar enlargement will inhibit the sphincter centre whilst it sets in action the detrusor. The other centres are the centre for the rectum (sphincter), the sexual centre, centre for the uterus, &c. A lesion destroying the portion of the lumbar enlargement in which the bladder and rectal centres are situated necessarily causes relaxation of the sphincters, so that the urine and fæces are

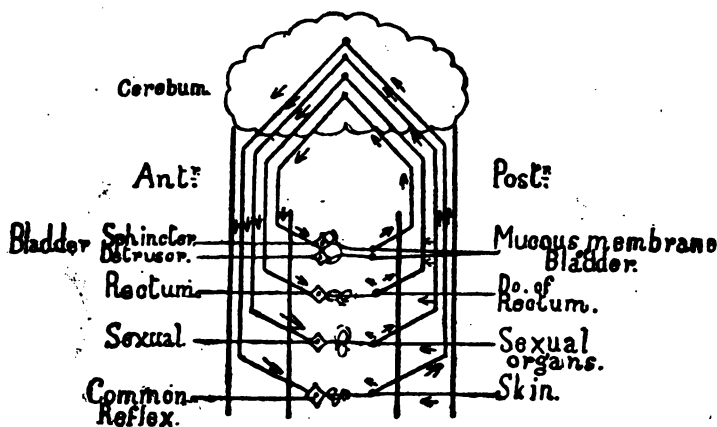


FIG. 31.

passed involuntarily as soon as they reach the relaxed sphincters. On the other hand, a lesion situated higher up the cord, which obstructs the tracts in which the volitional impulses descend, the rectal and bladder centres being intact, will simply interfere with the voluntary part of the act, constipation and retention of urine being generally observed in such cases.

CHAPTER XXIV.

THE SYMPTOMOLOGY OF CORD DISEASE.

As in the case of brain disease, cord affections are recognised by the derangements of the known functions of the organ, which, as we have seen, are reflex, trophic, vaso-motor, and the conduction of motor and sensory impulses. The symptoms of diseases of the spinal cord must therefore take the form of alteration of the reflexes; impairment of nutrition; vaso-motor phenomena; muscular derangements; and disturbances of sensation.

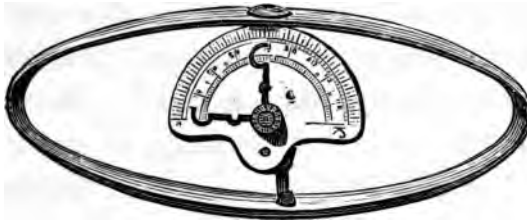
MUSCULAR DERANGEMENTS are discernible in the inability of the patient, however willing, to perform a required action at all, or to perform it in the natural way; or, secondly, as phenomena independent of the patient's volition or action. In the former case we meet with some form of paralysis, inco-ordination, or impaired muscular sense; in the latter, rigidity, spasms, or cramps.

Paralysis.—All degrees of motor weakness, from a slight paresis of one limb to complete paralysis of all four extremities, occur in cord disease. The lower limbs are much more frequently paralysed than the upper, and usually both legs are more

or less affected, constituting paraplegia. Spinal monoplegia is the paralysis of only one limb; spinal hemiplegia that of the arm and leg of the same side; cervical paraplegia, that of the upper extremities alone—a very rare condition.

To detect paralysis of the lower extremities the patient should be directed to perform certain movements free from, as well as in opposition to, resistance, the performance of which brings into play certain groups of muscles whose actions are known. Thus an attempt to walk will often reveal the distribution of the paralysis. It often happens that motor weakness confined to a limited number of muscles is not at once obvious to the physician, though the patient may insist that he is “weak in the legs;” in such a case the gait should be carefully observed, and a variety of motor performances, both when standing and lying, insisted on before declaring the patient free from paralysis. The following example will serve as an illustration:—A farm labourer came to me complaining of weakness of the left leg, which he stated was the result of a blow on the back and legs through a landslip. At first it was difficult to detect any paralysis, for, as he lay in bed, he appeared to perform with vigour all the gross movements required of him. When he walked the only peculiarity discoverable in his gait was his lifting the left leg higher than the right as

he advanced it. This he did to avoid scraping the toes along the floor, for paralysis of the *tibialis anticus* and *peroneus tertius* allowed the foot to drop. It was then observed that he was unable to flex the tarsus on the leg.



Dynamometer.

With the dynamometer the power of grasping with the fingers can be readily estimated. The mechanical and electrical irritability of the paralysed muscles should in all cases be carefully tested. (*See p. 56.*)

Inco-ordination and impairment of the muscular sense are also of frequent occurrence, and, like paralysis, are to be detected in the performance of volitional actions. In most of the cases of spinal disease inco-ordination is confined to the lower extremities, and is supposed to arise from the blocking in the posterior root-zones of the cord of the cerebello-afferent impressions which originate in the skin, muscles, tendons, joints, &c., of the lower extremities, and upon which depends the correct grouping of the muscular movements,

or accurate co-ordination. In consequence the patient performs movements in an awkward and jerking manner, and places great reliance on the aid of his sight to counteract his inability to co-ordinate. If the impairment be at all marked, he is unable to walk with his eyes closed, and in many cases finds it impossible to maintain his balance in the upright position, even with the eyes open and the feet separated. But nothing tries him so much as an attempt to stand in the so-called *ataxic position*—i.e., with the eyes shut and the feet close together.

Debove and Boudet* have recently revived Lockhart Clarke's view that the inco-ordination of *ataxics* depends on inequality of muscular tone. In explanation I would here remind the reader that a tonic muscle will reach its maximum contraction sooner than an atonic one, and hence when muscles are called into united action, some of which have a diminished, and others an increased or normal tone, an irregularity is manifested.

The muscular sense is not easy to test, especially in the lower extremities. When it is desired to test the arms it is useful to invite the patient to distinguish between the weights of different objects; it is well not to lay the test objects directly on the hand, but to suspend them from the limb or hand in a handkerchief.

* "Archives de Neurologie."

A rough way of testing the muscular sense of the arm is to require the patient to touch a prescribed part of his face—his nose, for example, with one finger, whilst his eyes are closed.

When the muscular sense of the lower extremities is impaired the patient is unable, whilst lying on his back with his eyes bandaged, to place his legs accurately in different positions when directed to do so; nor can he describe what position they are in when their attitude is altered by the observer.

The faradic current is, theoretically, the best test for the muscular sense; but in practice it is not found to be of much service except as a means of discovering muscular analgesia by inducing an artificial cramp.

Muscular Rigidity, Spasms, &c.—The tension or tonicity of the muscles is very often changed in cord disease, the alteration taking place either in the direction of increased or diminished tonicity. When increased, the condition is one of rigidity; the muscles are then firm and more or less rigid, and the limb is generally maintained in an extended position. Flaccidity of the muscles, the result of diminished tonicity, is also frequently observed.

DISTURBANCES OF SENSIBILITY.

Impairment of the sensory functions of the skin is shown by objective disturbances of touch, pain,

250 *Mode of Testing Cutaneous Sensibility.*

and temperature, and by subjective sensations, or paræsthesiæ.

Anæsthesia, or the loss of tactile sensibility, though a common phenomenon, is not so frequently met with as analgesia, or the loss of the sense of pain whilst touch is preserved.

Pressure and tickling are so closely associated with touch as to render their special investigation unnecessary.

To test tactile sensibility the skin should be touched lightly in different places with a blunt-pointed pencil, the patient being invited to state accurately, with his eyes closed, where the contacts are made, or to distinguish between the applications of the pencil when it is alternately pressed firmly and lightly against the skin. He should also be required, with his eyes shut, to give a description of a small object such as a coin or key put into his hand.

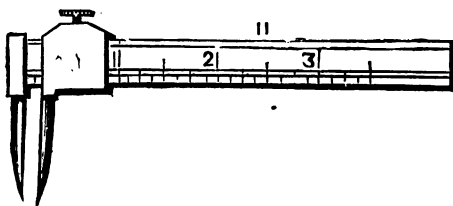


FIG. 32.—Sieveking's *Æsthesiometer*, 10 centimeters long and graduated to millimeters.

The æsthesiometer (fig. 32) is often employed in testing the sense of touch, but it furnishes very unsatisfactory results, and as a clinical

instrument might almost be abandoned in its present form.

Pain may be tested by pricking with a pin, which should be applied somewhat sharply, as gradual insertion is scarcely a sufficient test. The faradic current applied to the skin with dry rheophores is also a good test, care being taken that the skin is perfectly dry, otherwise the current will penetrate deeply and affect the muscles.

To test thermal sensibility a small metallic spoon, heated or cooled in hot or cold water, may be used: it should be recollected that the stimulus must be either hotter or colder than the skin before it can excite either a hot or a cold sensation.

Hyperæsthesia of the skin is also of frequent occurrence; it is detected without difficulty, for when it is present a tactile impression is usually magnified into a painful one.

The subjective sensations, or *paræsthesiæ*, complained of by patients suffering from disease of the spinal cord, are numbness, "needles and pins," formication, or sense of creeping, furry sensation (generally under the feet), and the girdle sensation (the sensation of a girdle tied round the body), &c.

The reflexes are most important features in spinal diseases, and should be carefully investigated in every case. The principal of the common skin reflexes are:—the plantar, shown by jerking of the leg when the sole of the foot is

tickled; the cremasteric, shown by drawing up of the testicle when the adjacent part of the inner surface of the thigh is irritated; the abdominal, obtained by irritating the skin of the lateral surface of the abdomen, when the abdominal muscles contract; the epigastric, shown by contraction of the muscles of the epigastrium on irritation of the skin of the sides of the chest beneath the nipples.

The plantar, which is related to the lower part of the lumbar enlargement of the cord, is generally more or less present in health, and may be quite abolished, masked, or increased in disease.

The cremasteric, though generally well marked in children, is often absent in adults in health, and is related to the first and second lumbar nerves. The abdominal is generally present to a certain, though slight, extent, in health; it corresponds to the lower dorsal nerves from the eighth to the twelfth. The epigastric is often absent in health, and is related to the fourth, fifth, and sixth dorsal nerves.

The *knee-jerk*, so-called patellar-tendon reflex, and *ankle-clonus*, must also be carefully investigated. The former is elicited by striking sharply, but without undue force, the ligamentum patellæ, when the foot and leg are jerked forward from contraction of the quadriceps femoris. The latter is a rhythmical jerking of the foot from jerking of

the muscles of the calf, obtained when the tendo-Achilles is suddenly put on the stretch by flexing the foot rapidly on the leg. The *knee-jerk* is best obtained by placing the patient on his back, supporting the leg in a semi-flexed position with the left hand, and percussing the tendon with one finger of the right. Then, to elicit *ankle-clonus*, the patient's leg being still supported by the left hand, the foot is suddenly flexed with the right.

It is common enough in cases in which the *tendon-jerk* is markedly increased, especially in descending degeneration of the lateral columns of the cord, to observe it in the upper extremities as well. The biceps tendon, for example, furnishes it occasionally, and sudden extension of contracted fingers will sometimes, by putting the tendons on the stretch, be followed by rhythmical jerking of the fingers.

As the sphincters of the rectum and bladder are under reflex control of the lumbar enlargement of the cord, disease very often causes marked disturbances of defecation and micturition, which are spoken of as impairment of the *rectal* and *vesical reflexes*.

Trophic alterations in the joints and skin, resulting in disorganisation of the joints and bed-sores, are met with from time to time. Œdema, redness of the skin, and increased or diminished temperature, are noted as the result of vaso-motor changes.

CHAPTER XXV.

SECONDARY DEGENERATIONS.

IN the following brief allusion to the secondary degenerations, reference is made to the sclerotic changes to which certain tracts in the brain and cord are liable whenever the continuity of their fibres is interrupted by a lesion in any part of their course, whether that lesion be situated in the cortex of the brain, internal capsule, crura, pons, medulla, or cord. The cerebral tracts ordinarily degenerated are the fibres which occupy the middle third of the posterior division of the internal capsule, the middle third of the crusta of the peduncles, and the anterior pyramids of the medulla. As they enter the cord the majority of the degenerated fibres decussate, and occupy the posterior part of the lateral column of the opposite side (crossed pyramidal tract), whilst the smaller division descends the anterior column of the same side, and occupies its internal aspect (column of Türck). The other tracts liable to degeneration in the cord are the columns of Goll, and the direct cerebellar tracts.

Secondary degeneration is either *ascending* or

descending, and always takes place in the direction from the trophic centre of the fibre towards its distal end. Thus, in the case of the columns of Goll, or the postero-internal columns of the cord, and the direct cerebellar tracts, the trophic centres for the fibres which compose the former being situated low down in the posterior gray horns of the cord, and those for the latter in the vesicular column of Clarke, the degeneration will ascend the cord, and is consequently described as

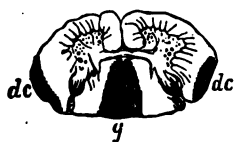


FIG. 33.



FIG. 34.



FIG. 35.

ascending degeneration (figs. 33, 34, and 35).* But, on the other hand, as it is probable that the trophic centres for the crossed and direct pyramidal tracts are situated in the gray matter of the cortex of the brain, their fibres will necessarily degenerate



FIG. 36.



FIG. 37.



FIG. 38.

downwards, constituting *descending degeneration* (figs. 36, 37, and 38).

* *dc* indicates the direct cerebellar tracts ; and *g*, the columns of Goll.

The nature of the degeneration is not precisely known, though it is generally believed to be a

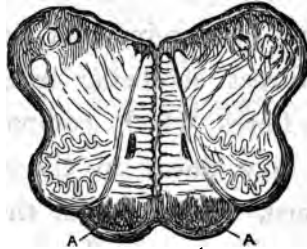


FIG. 39 (after CHARCOT).—*Transverse section of the Medulla on a level with the middle of the olivary body.*—A, A, descending degeneration of the anterior pyramids.

simple atrophic change from impaired nutrition, and not, as some have suggested, a direct spreading of an irritative lesion along the fibres from the primary focus of disease. According to recent researches the atrophic change or degeneration commences about fourteen days after the establishment of the interruption in the fibres, and takes from four to six weeks or more to become prominently developed.

Some lesions of the brain appear to be more commonly followed by *descending degeneration* than others. Hæmorrhages are especially apt to induce it in an exaggerated degree, tumours also, when they encroach upon the internal capsule, generally give rise to it, but embolic softening is less liable to do so.

Rigidity of the muscles, with contractures of the limbs, and increased tendon-jerk, are the principal phenomena that attend *descending degenerations*.

CHAPTER XXVI.

DISEASES OF THE DURA MATER.

PACHYMENINGITIS SPINALIS

(Inflammation of the Spinal Dura Mater).

WE meet with two forms of this affection, Pachymeningitis Externa and Pachymeningitis Interna, according as the external or internal surface of the dura mater is attacked by the morbid process.

PACHYMENINGITIS EXTERNA.

External pachymeningitis is a very important condition, and its significance has been rendered much more prominent by the part which Michaud and Charcot have assigned to it in the production of paraplegia in Pott's curvature. The lesion consists of inflammation of the external surface of the dura mater and the adjacent cellular tissue. The result of this inflammatory process, which is very often secondary to caries of the bodies of the vertebræ, is a more or less chronic deposit on, or exudation from, the surface of the mem-

brane of a "purulent, plastic, or tuberculous material" (Erb).

The deposit, though seldom extending in a longitudinal direction beyond two or three vertebræ, is often so thick as to cause marked compression of the cord itself. The nerve-roots which pass through the mass are likewise subjected to pressure, and in consequence become the seat of prominent lesions. In many cases myelitis, softening, and secondary degenerations arise from the cord.

As already stated, vertebral caries is the most frequent cause of the disease. In other cases the inflammation is set up by pus finding its way into the spinal canal from abscesses located near the spinal column.

SYMPTOMS.

The affection is usually attended by pain in the back referred to the situation of the lesion, and a *girdle* pain, with shooting pains in the limbs, and hyperæsthesia of the skin. Stiffness or rigidity of the spinal muscles is also a common phenomenon.

Subsequently the symptoms of compression of the cord make their appearance. (*See* p. 297.)

The diagnosis, apart from vertebral caries, is very difficult, as the symptoms resemble other forms of spinal meningitis; indeed, the clinical

recognition of the lesion may be said to hang upon the discovery of caries.

The prognosis is upon the whole favourable in cases arising from Pott's disease, but in all instances an opinion as to the result must be founded upon the nature of the primary lesion.

The treatment, like the prognosis, depends upon the cause of the attack. The first aim should be the removal or relief of the original affection. Counter-irritation, either in the form of small blisters frequently applied, or the cautery, is usually of much service.

PACHYMENINGITIS INTERNA.

Two varieties of inflammation of the internal surface of the dura mater have been described, Hypertrophic Pachymeningitis, or "*Pachyménigite Cervicale Hypertrophique*" (Charcot), and Hæmorrhagic Pachymeningitis.

Hypertrophic pachymeningitis consists of chronic inflammatory thickening, usually annular in form, of the dura mater in the cervical region. The pia mater and arachnoid nearly always share in the thickening, and are found adherent to the dura mater. The nerve-roots arising from the cord at the situation of the lesion are atrophied from pressure, and the cord itself is subjected to slow compression. This affection is

said to arise from exposure to cold, and from alcoholic excesses.

The symptoms which characterise the first few months consist of violent neuralgic pains in the neck and upper extremities, girdle pain round the thorax, hyperæsthesia of the skin, and rigidity and spasms of the muscles supplied by the nerves which pass through the thickened membranes. Later on atrophy and paralysis with anæsthesia follow, causing contractures with deformities. Subsequently the lower extremities become affected, for myelitis and secondary descending degeneration are wont to ensue in the compressed cord.

Some cases show a remarkable tendency to recovery after a prolonged stationary period, and almost complete restoration of motor power may result; but in the majority the paralysis and deformities, especially of the upper extremities, are permanent.

The treatment lies in the direction of counter-irritation, with the application of the continuous current to the spine and paralysed muscles. Sometimes iodide of potassium appears to do good.

Hæmorrhagic pachymeningitis is nearly always associated with a similar affection of the cerebral dura mater. The internal surface of the dura mater is found covered with an exudation of a rusty-brown colour, composed of fibrine and a

delicate connective tissue, and studded with numerous minute hæmorrhages.

The symptoms of this disease are very obscure, and most commonly make their appearance along with cerebral symptoms. In the main the clinical features are those of a chronic meningeal affection—pain and stiffness in the back, pains and motor weakness in the limbs, areas of hyperæsthesia and anæsthesia of the skin, added to which are occasional attacks suggestive of a sudden meningeal hæmorrhage.

In attempting a diagnosis great prominence should be given to the coexistence of ill-defined spinal meningeal symptoms, and symptoms of hæmatoma of the cerebral dura mater. (*See* p. 117.)

CHAPTER XXVII.

DISEASES OF THE PIA MATER.

LEPTOMENINGITIS SPINALIS

(Inflammation of the Spinal Pia Mater and Arachnoid).

THE great majority of cases of spinal meningitis consist of inflammation of the pia mater and arachnoid; in comparatively few instances does the morbid process attack the dura mater.

The affection is either acute or chronic.

ACUTE MENINGITIS.

MORBID ANATOMY.

After an initial period characterised by congestion, in which the cord shares to a considerable extent, the pia mater and arachnoid become opaque and swollen, and covered with fibrinous or purulent exudation, and the spinal fluid turns turbid. The exudation is rarely circumscribed, being generally spread over a considerable area of the cord. The membranes on the posterior surface are commonly most affected, and the nerve-roots are always more or less involved. The inflammatory process is often associated with a similar condition within the

cranium, but the medulla is seldom the seat of abundant exudation. The cord may present morbid changes; in some cases it is congested, cedematous, and softened.

As the condition becomes chronic, the inflammatory products are transformed into thickenings, and adhesions take place; changes of a sclerotic and atrophic character then ensue in the cord, and the nerve-roots become wasted.

ETIOLOGY.

Spinal meningitis attacks children and young people, and the male sex most frequently. Exposure to wet and cold and to the extremes of temperature has often been known to excite the disease. Injuries, caries of the vertebræ, and other surgical lesions, as well as acute rheumatism, tuberculosis, pneumonia, diphtheria, small-pox, and typhoid fever, stand in an etiological relation to acute meningitis.

SYMPTOMS.

It is not often that we have to do with an attack of acute spinal meningitis simply; for, owing to the frequent participation of the cerebral pia mater in the spinal changes, the aspect of the case is commonly a complex one.

The onset is generally sudden, a prodromal stage showing itself with comparative rarity.

An initial rigour is followed by a state of pyrexia, marked by high temperature and a rapid, full, and hard pulse. Intense pain in the back, increased by every movement of the spine and limbs, is an early symptom. The muscles of the back are stiff; in some cases the head is drawn backwards, in others the whole column is arched as in tetanus. The limbs are also frequently affected with spasm, so that the knees may be bent, and the thighs firmly flexed on the abdomen.

The *reflexes* are increased during the irritative stage, but as the case advances and paralysis gives place to spasm they are either lost or markedly diminished. The period of irritation is attended, in addition to pain in the back and muscular spasm, with shooting pains in the extremities and hyperæsthesia of the skin. Spasm of the sphincters occasionally occurs, producing retention of urine and constipation, and the breathing may be interfered with from spasm of the respiratory muscles.

Should the case survive the acute, and pass into the chronic, stage, areas of anæsthesia, motor paralysis, atrophy, and contractures make their appearance.

DIAGNOSIS, COURSE, AND PROGNOSIS.

As a rule the clinical phenomena of acute spinal meningitis can easily be recognised by the

acute onset, feverish symptoms, pain and rigidity of the back, with spasms of the extremities and hyperæsthesia which form a symptom-picture not readily mistaken.

In cerebro-spinal cases headache, vomiting, loss of consciousness, and other cerebral symptoms are added to the features of the spinal lesion.

In tetanus, which may be confounded with acute meningitis, there are neither cerebral symptoms, nor cutaneous hyperæsthesia, whilst trismus is a prominent feature, and there is commonly a history of a wound.

The prognosis is very unfavourable in the most acute cases, especially in the tubercular form of the disease (tubercular cerebro-spinal meningitis). But all cases must be regarded as serious, for should the patient even survive the acute stage the after effects are often most disastrous. In some cases death takes place within a day or two from the commencement, but it often happens that the condition passes into one of chronic spinal meningitis. The most favourable varieties of the affection are the rheumatic and syphilitic.

TREATMENT.

When the case is seen early an attempt may be made to arrest the inflammation by the use of the ice-bag, local blood-letting, and a good purgative. Morphia, chloral, and bromide of potassium may

be employed to relieve the pain and spasms; subsequently, blisters or mercurial inunction to the back may prove useful, and iodide of potassium is a valuable internal remedy. All attempts to treat the resulting paralysis by electricity should be deferred until the irritative stage has quite passed away.

CHRONIC MENINGITIS.

Chronic meningitis may be met with as a late stage of an acute affection, or as a chronic inflammation from the first.

ETIOLOGY.

The causes of acute meningitis must be regarded as standing also in a causal relation to chronic inflammation of the meninges. In addition, it may be traced to new-growths of the vertebræ or membranes, abuse of alcohol, sexual excesses, the extension of chronic inflammatory processes from the substance of the cord (sclerosis, myelitis, &c.), or syphilis.

MORBID ANATOMY.

More or less congestion, with thickening of the pia mater and arachnoid, commonly limited in its distribution except in cases resulting from acute

inflammation, are its characteristic pathological features. The cord is usually the seat of secondary lesions of the nature of myelitis and sclerosis.

SYMPTOMS.

When the inflammatory process is chronic from the beginning the onset is generally very insidious, and, indeed, may be latent for some time. The symptoms may be said to arise from the nerve-roots becoming involved in their passage through the diseased membranes. At first subjective sensations (*paræsthesiæ*) are complained of in the limbs. The skin presents hyperæsthetic areas, and patches of anæsthesia make their appearance. There is often a vague, dragging pain in the back, intensified by every movement of the spine. The girdle sensation is common, and shooting pains in the limbs are especially frequent.

The motor roots nearly always suffer. At first there may be stiffness of the muscles of the back and neck, and drawing up of the limbs, but later on paralysis and atrophy of the muscles ensue.

DIAGNOSIS.

Locomotor ataxia and progressive muscular atrophy are the principal conditions with which chronic spinal meningitis may be confounded. From the former it is distinguished by the paralysis and atrophy, and absence of ataxia and inco-ordina-

tion; from the latter, by the irregular distribution of the symptoms, and presence of hyperæsthetic and anæsthetic areas, and spinal pain.

The prognosis is always grave, for more or less permanent disablement may be looked for.

TREATMENT.

Counter-irritation along the spine is useful. Iodide of potassium and cod-liver oil occasionally do good, and mercury is essential in syphilitic cases. The continuous current to the spine and paralysed muscles deserves a trial.

CHAPTER XXVIII.

HYPERÆMIA AND ANÆMIA OF THE
CORD.

HYPERÆMIA.

IN the present state of our knowledge very little can be said respecting congestion of the cord as an unequivocal cause of symptoms of disease; moreover it is impossible to discriminate clinically, between hyperæmia of the substance of the organ and that of its envelopes.

The morbid anatomy is extremely unsatisfactory, owing in great measure to post-mortem accumulation of blood in the vessels due to the ordinary position of the body, and because of the tendencies of the arteries in contracting after death to empty themselves into the veins.

In active congestion the vessels of the pia mater are full and red, and fresh sections of the cord are rose-colour, the gray matter being darker than normal; and minute ecchymoses are often seen dotted through the substance of the cord. In passive hyperæmia the membranes and cord present a cyanotic aspect, the veins are tortuous and full, and the spinal fluid is generally some-

what increased. It is commonly stated that the membranes become thickened, and the vessels permanently dilated and varicose in chronic cases.*

Violent exercise, exposure to intense cold, sexual excesses, abuse of alcoholic stimulants, strychnia poisoning, and the suppression of customary discharges, such as menstrual or hæmorrhoidal flux, are the most frequent causes of spinal congestion.

SYMPTOMS.

Foremost amongst the symptoms may be mentioned a dull, aching pain in the back, usually in the lumbar or sacral regions. Dragging pains in the limbs, subjective sensations, such as "needles and pins," heaviness and weakness in the legs, with a sense of fatigue on exertion are also observed, but complete paraplegia never.

The phenomena are usually more pronounced in the morning, owing to the assumption of the recumbent position during the night; hence the patient is more paralysed in the morning before rising from bed than in the evening before he retires (Hammond).

In simple hyperæmia vesical symptoms are very rarely, and bed-sores never, met with, and there is an absence of febrile disturbance. According to Erb and Rosenthal the reflexes are somewhat increased;

* Rosenthal ("Diseases of the Nervous System," vol. i., p. 178) regards chronic hyperæmia as a cause of softening of the cord.

but Hammond states that they are invariably lessened. Rosenthal (*op. cit.*) has seen increased electrical excitability.

The prognosis is usually favourable. Sometimes recovery takes place almost suddenly, owing to the re-establishment of an habitual bleeding, menstrual or hæmorrhoidal. On the other hand, relapses are common, and chronic changes may be set up which occasion permanent disturbances.

The treatment obviously depends upon the cause of the hyperæmia. In cases due to the suppression of a customary discharge, leeches to the cervix uteri or anus, as the case may be, are useful; or the spinal column may be leeches. The application of cold to the spine by means of Chapman's ice-bag, or better still by Leiter's pliable metal temperature regulators (fig. 40), should be tried. Ergot



FIG. 40.

and belladonna, in doses of a drachm of the former (liquid extract), and 10 mins. of the latter (tinct.), three times daily, will be found to exercise a most salutary effect by contracting the blood-vessels. The diet should be carefully regulated, and attention given to the action of the bowels.

ANÆMIA.

In spinal anæmia the anæmic portions are pale and bloodless, the pallor being especially noticeable in the gray substance; the membranes are also observed to be pale and the minute vessels entirely empty. In some old-standing cases softening makes its appearance, in others the consistence of the cord is increased.

The anæmia, of course, depends upon a lessened blood-supply which may have its origin in vaso-motor spasm, weak heart, severe hæmorrhages, diminution in the calibre of spinal vessels as in congenital narrowing, atheroma, or arterio-capillary fibrosis (as in granular kidney), or in interference with the circulation due to an arrest outside the spinal canal, such as occlusion of the abdominal aorta.

Vaso-motor disturbances as a cause of spinal anæmia usually operate in the female sex. The so-called *reflex paralyses* are supposed to be due to vaso-motor spasm, but in my opinion the rare cases of paraplegia arising from renal, vesical, or uterine irritation depend upon inhibition of the motor cells. Softening resulting from occlusion of a spinal vessel is generally situated very low down in the cord, a fact which Dr. Moxon has shown to be due to the peculiarity of the circulation of that portion.

SYMPTOMS.

Motor paralysis, usually confined to the lower extremities and seldom well marked, tremor on exertion, backache, tenderness on pressure over certain points of the spine, hyperæsthesia of the skin and paræsthesiæ ("needles and pins," &c.), and increased reflexes are the usual phenomena of spinal anæmia. The symptoms are generally more pronounced after the patient has been going about for a few hours, and are somewhat relieved on lying down. The sphincters are very rarely affected.

In acute cases the diagnosis of spinal anæmia suggests itself on the discovery of the above symptoms, more or less pronounced, associated with a sudden loss of blood, or occlusion of the abdominal aorta, as in the case of an aneurism. The chronic forms are with difficulty distinguished from certain organic lesions, such as chronic myelitis, &c.; but the amenability of the affection to treatment, and the recognition of an existing cause of anæmia should prevent a mistake.

The treatment consists in the removal of the causes of the disease, keeping the patient in a lying posture, the administration of iron, strychnia, and phosphorus, and the application of warmth and the constant current to the spine.

CHAPTER XXIX.

SPINAL HÆMORRHAGE.

HÆMATORRHACHIS

(Meningeal Hæmorrhage).

EXTRA-MEDULLARY hæmorrhage of the cord, though uncommon, is a recognised affection.

MORBID ANATOMY.

The extravasation occurs most frequently into the connective tissue between the vertebræ and the external surface of the dura mater. It is also found occasionally between the dura mater and the arachnoid, and sometimes, though very rarely, beneath the latter. The effused blood is generally black and coagulated, though it may be fluid and mixed with clots. The extravasation may embrace the whole cord external to the membranes, but is commonly much more limited; not unfrequently the clots surround the roots of the nerves as they pass out of the vertebral canal. In a longitudinal direction the clot very rarely extends more than an inch or two along the surface of the cord. The extravasations may compress the cord and induce softening in it, though it is only the larger ones that do so.

ETIOLOGY.

The commonest cause of spinal meningeal hæmorrhage is injury to the spinal column; severe blows or falls, with or without direct laceration of the membranes, frequently induce it. Inflammatory affections of the vertebræ and membranes also occasionally lead to extravasation. Amongst other causes may be mentioned the rupture of vessels during the plethora attending the sudden arrest of menstrual or hæmorrhoidal flux, eclampsia, epilepsy, violent exercise, tetanus, &c., when it is most probable that arterial degeneration precedes the rupture. Sometimes the effused blood finds its way from the skull into the spinal canal. Purpura, small-pox, typhoid fever, and other acute diseases have been known to occasion hæmorrhage in some few cases. And, lastly, a thoracic or abdominal aneurism may open into the canal.

SYMPTOMS.

In the majority of cases the effusion is a sudden occurrence, and is attended by a sharp pain in the back, and a certain degree of spinal paralysis; sometimes, but rarely, consciousness is lost, a symptom indicative of co-existing cerebral lesion. After the onset, in addition to pain in the back and shooting pains in the limbs and parts supplied by the nerves whose roots are affected, and hyper-

æsthesia, stiffness of those muscles of the vertebral column and limbs which are related to the affected spinal region becomes a prominent feature. Along with this tonic spasm, spasmodic jerking of the muscles is often present.

Subsequently anæsthesia—generally occurring in patches and limited in its distribution, and motor weakness make their appearance, but the paralysis is seldom very pronounced. The situation and extent of the extravasation will in a great measure regulate the clinical aspect of the case. In extra-medullary hæmorrhage of the cervical portion of the cord, for example, in addition to the pain and stiffness of the muscles of the shoulders, neck, and arms, it is common to meet with dilated pupils, difficulty of swallowing, and interference with the respiratory functions. When the lower portion of the cord is attacked, usually from a fall or blow upon the loins, in addition to the symptoms already referred to, there may be retention of urine and constipation, as well as priapism.

PROGNOSIS.

The majority of mild cases appear to recover, though a stage of inflammatory reaction is generally passed through before the improvement is observed. In severe cases the prognosis is always unfavourable, especially when the upper

part of the cord is the seat of the lesion. In some cases the fatal result is hurried by myelitis, bed-sores, cystitis, &c.

TREATMENT.

Absolute rest must be insisted upon. Ice may be applied to the spine, and hypodermic injections of ergotin and sulphate of atropia employed, the following prescription answering very well for the purpose:—

° R. Atrop. Sulp. ($\frac{1}{2}$ gr.).
Ergotin (2 drs.).
Chloral. Hyd. (10 grs.).
Aquæ destil. (to 1 oz.).
M. fiat injec. hypoderm.

Twelve drops to be injected deep into the muscles once or twice daily. After the inflammatory period has entirely subsided, the paralysed muscles should be faradised.

HÆMATOMYELIA—SPINAL APOPLEXY

(Hæmorrhage into the Substance of the Cord).

ETIOLOGY AND MORBID ANATOMY.

Intra-medullary hæmorrhages are rare, and very seldom occur as spontaneous extravasations independent of previous changes in the substance of the cord.

* Each dose contains three grains of ergotin and the eightieth of a grain of sulphate of atropia.

The morbid conditions which most frequently underlie spinal hæmorrhages are myelitis,* progressive muscular atrophy, changes in the vascular walls of the nature of fatty degeneration and aneurismal dilatation, and tumours. Amongst the immediate causes of the rupture of the vessel, when the predisposing morbid conditions exist, may be mentioned exposure to cold, sexual excesses, injuries, and, in general, any condition calculated to induce active hyperæmia of the cord. Spinal apoplexy, unlike cerebral hæmorrhage, occurs more frequently in early and middle life (10—40) than in old age. Men are more frequently attacked than women. The extravasation is nearly always confined to the central gray matter, and the clot is usually small and circumscribed; in some cases, however, the hæmorrhage is diffused almost throughout the whole length of the gray axis, a condition usually the result of hæmorrhagic myelitis. Inflammatory changes attended by softening are commonly found in the neighbourhood of the clot.

SYMPTOMS AND DIAGNOSIS.

It is usual to meet with symptoms indicative of spinal hyperæmia—backache, numbness in the limbs, feeling of fatigue after exertion, &c.—for

* According to Hayem, hæmorrhage into the tissue of the cord only occurs when it has been previously inflamed.

some time before the onset of the hæmorrhage. The extravasation, as a rule, *reveals itself all at once* by a sudden paralysis, generally of a paralytic type, and severe pain in the back. The paralysed limbs are found to be anæsthetic, the muscles are flaccid, the sphincters affected, and the reflexes, at first abolished owing to shock, subsequently may be increased for a time, but later on are wont to disappear. When the lower part of the cord is the seat of the hæmorrhage the reflexes remain absent from the first, and the muscles atrophy and lose their electrical excitability. At times secondary descending degenerations occur resulting in rigidity of the limbs. Consciousness is very rarely lost at the onset, thus distinguishing spinal from cerebral hæmorrhage; occasionally, however, when an abundant extravasation takes place in the cervical region, a temporary loss of consciousness ensues. Bed-sores and cystitis are frequent and serious complications.

Hæmorrhage into the cord may be confounded with meningeal hæmorrhage, acute anterior poliomyelitis or infantile paralysis, and acute central myelitis.

In meningeal hæmorrhage the phenomena have more of an irritative than a paralytic character, *i.e.*, motor spasms and hyperæsthesia are more prominent than paralysis and anæsthesia.

The symptoms of a case of acute central myelitis

are longer in developing than those of spinal apoplexy, and the lesion in the former affection shows a tendency to ascend, whilst in the latter it is stationary.

Infantile paralysis may easily be distinguished from hæmorrhage by recollecting that the former is nearly always ushered in by a feverish attack, and, further, that in anterior polio-myelitis there are neither sensory nor vesical disturbances.

The prognosis is, generally speaking, unfavourable; a hæmorrhage into the upper cervical region is rapidly fatal, owing to respiratory complications.

TREATMENT.

When the opportunity presents itself, prophylactic measures should be adopted with the view of obviating the tendency to hyperæmia and inflammation. After the occurrence of the hæmorrhage little can be suggested beyond the use of the spinal ice-bag, complete rest, and ergot with bromide of potassium.

CHAPTER XXX.

MYELITIS

(Inflammation of the Cord).

ACUTE MYELITIS.

IN this section it is only my intention to consider the subject of acute myelitis so far as it relates to inflammation of the cord in general, reserving for subsequent consideration those acute lesions which are confined to special physiological systems of the cord, as, for example, polio-myelitis anterior acuta.

MORBID ANATOMY.

The extent and situation of the inflammatory process vary very considerably in different cases. Having regard to these variations, authorities describe several forms of acute myelitis, viz.:—

1. *General*, in which the whole, or nearly the whole extent of the cord is involved in the morbid process.
2. *Central*, in which the central gray matter is principally involved.
3. *Transverse*, in which the process is limited longitudinally, but occupies the whole transverse section.
4. *Bulbar*, signifying that the medulla is likewise involved.

5. *Disseminated*, denoting the occurrence of small foci of myelitis scattered through the cord. 6. *Unilateral*, in which the lesion is limited to a lateral half of the cord, or a part thereof.

The affected part passes first through a stage of redness and congestion which is characterised microscopically by distended blood vessels, escape of leucocytes into the lymphatic sheaths, swelling of the glia cells of the interstitial substance, and hypertrophy of the axis cylinders and nerve cells. Then follows the stage of softening and disorganisation in which the nerve elements are seen to be broken down, and oil globules from fatty degeneration of the medullary sheaths appear in abundance. Subsequently the fluid portions of the softened mass are absorbed, and nothing is left but the vascular and connective-tissue network. This usually results in the formation of a cicatrix, or the development of a cyst.

ETIOLOGY.

The causes of acute myelitis are by no means thoroughly understood. The following influences are, however, supposed to be capable of exciting the disease:—exposure to extremes of temperature, especially cold; injuries, such as a blow, a crush, or incised wounds; the extension of inflammation from neighbouring parts, as the bodies of the vertebræ and meninges; acute exanthemata,

diphtheria, and other acute inflammatory affections; excessive muscular exertion; venereal excesses, &c.

SYMPTOMS.

In no other spinal lesion are the clinical phenomena so protean as in myelitis. The symptoms vary so much in the different cases as to make it quite impossible to attempt a description suitable to the size of this work and yet sufficiently comprehensive to include in their kaleidoscopic combinations all the features of the disease. The wide differences observed in cases of myelitis are easily accounted for by the want of uniformity in the acuteness of the process, and the situation and extent of the disease.

The majority of the attacks commence with a certain degree of febrile disturbance, pain in the back, and tingling or numbness in the extremities. Occasionally a well-marked rigour ushers in the case, and high fever precedes the distinct spinal symptoms. After this premonitory stage the spinal lesion reveals itself in the first instance by symptoms of irritation, and subsequently by paralysis. The symptoms of irritation are pains, sometimes of the girdle description, and sometimes sharp and shooting, hyperæsthesia of the skin and muscles, and muscular twitchings or cramps.

In many cases motor weakness comes on with

great rapidity; in the most acute, an hour or two suffices for the full development of the paralysis. In others, again, the irritative phenomena gradually give place to paresis which may or may not advance to complete paralysis. The sensory impairment commonly takes the form of anæsthesia; at first with a certain amount of hyperæsthesia as regards pain, but subsequently analgesia (loss of painful impressions) accompanies the anæsthesia. Bed-sores often form over the sacrum, trochanters, or heels; and the rectal and vesical functions are frequently deranged; whilst acute cystitis and pyelo-nephritis are not unfrequent complications. The form and distribution of the paralysis, sensory and motor, must of course depend upon the situation and extent of the lesion.

In *Acute General Myelitis* there is complete anæsthesia and motor paralysis of the legs, with flaccidity of the muscles, and loss of power over the bladder and rectum. The reflexes are abolished, bed-sores form, the paralysed muscles atrophy, and the morbid process shows a disposition to ascend the cord, affecting the upper extremities and respiratory muscles. Cases of *acute central myelitis* run a very similar course.

In *Acute Transverse Myelitis* the clinical picture varies according as the lesion occupies the dorso-lumbar, the dorsal, or the cervical regions. This variety most frequently follows very severe trau-

matic injuries of the cord. When the lesion is situated low down in the lumbar enlargement, the jerking of the limbs and other irritative phenomena soon give place to paralysis, and complete paraplegia with flaccidity of the muscles, loss of power over the bladder, and total abolition of the reflexes become early established, whilst bed-sores are seldom absent. When the transverse lesion is situated in the dorsal region secondary descending degenerations occur attended by rigidity of the paralysed muscles and increased reflexes, especially the various tendon-jerk phenomena. Further, there is generally a difficulty in micturition, but no sphincter paralysis, and it is very rare to meet with bed-sores. When the cervical region is attacked there is pain and stiffness in the back of the neck, and paralysis of the upper extremities.

Acute Bulbar Myelitis is characterised by a sudden onset, headache, vomiting, and difficulty in swallowing. Disturbances of respiration, with paralysis of the tongue, soft palate, and the lower muscles of the face, make their appearance early. In addition the limbs are observed to become paralysed, and there is a marked tendency to the formation of bed-sores.

In *Acute Disseminated Myelitis* the symptoms are extremely variable; but there is generally paraplegia with vesical paralysis.

Acute Unilateral Myelitis is marked by a rapidly developed lesion which gives rise to motor paralysis, increased temperature, and hyperæsthesia on the affected side, and incomplete anæsthesia on the opposite side.

DIAGNOSIS.

The diagnosis of acute myelitis is rarely attended with much difficulty; for the clinical picture of a spinal lesion ushered in by febrile disturbances, a certain degree of pain, and motor spasms, speedily followed by motor and sensory paralysis, weakness of the bladder, and bed-sores, is almost pathognomonic. But inasmuch as the symptoms are not always grouped in this characteristic manner, it will be necessary to attend to a few features which serve to distinguish acute myelitis from some other affections liable to be confounded with it. In *acute meningitis* the symptoms of irritation (pain and motor spasms) are much more pronounced than in myelitis, whilst the paralytic features of the former are comparatively slight, and bed-sores seldom make their appearance. But it must not be forgotten that in many instances the two affections are combined.

Cases of *hæmatomyelia* or *spinal apoplexy* may resemble acute myelitis very closely; a sudden attack resulting at once in complete paraplegia

without any antecedent febrile disturbance, is strongly indicative of a simple hæmorrhage.

Frequently it is almost impossible to distinguish Landry's, or acute ascending, paralysis from acute central myelitis, but the absence of phenomena of irritation and of sensory disturbances in the case of the former, along with its progressive character, should aid in forming an opinion.

Hysterical paralysis as distinguished from acute myelitis is usually characterised at the onset by rigidity of the muscles, pronounced analgesia of the skin, and increased tendon-jerk phenomena; and, further, the functional attack is never accompanied by bed-sores or sphincter paralysis.

COURSE AND PROGNOSIS.

Cases in which less than a week is occupied by the development of the disease are accurately described as acute, but the most severe develop in a few hours or less. When the cervical portion of the cord is the seat of an acute inflammation death often takes place within a few days from the onset. Cases in which the lumbar enlargement is involved not unfrequently become chronic, unless a fatal result should ensue from bed-sores and acute cystitis. On the whole acute myelitis must be regarded as a most serious affection;

circumscribed traumatic lesions are the most favourable, and acute central myelitis and hæmato-myelitis are the least so. Although some few cases run a fortunate course, and marked improvement up to a certain point may be observed, it must be borne in mind that complete recovery is almost unknown.*

TREATMENT.

Therapeutics have hitherto been found of little avail in this disease. Besides the administration of ergot and belladonna, and the use of the spinal ice-bag, little can be done in the acute stage further than to guard against bed-sores by putting the patient on a water bed and sponging the parts exposed to pressure with whiskey lotion. Two cases of acute myelitis recently under my care treated with hypodermic injections of ergotin and atropia have done remarkably well.

After two or three weeks have elapsed counter-irritation and galvanism may be employed to the spine (Hammond recommends the actual cautery), along with the internal administration of iodide of potassium and the tribasic phosphate of silver, the former in a mixture, and the latter in gr. $\frac{1}{4}$ pill three times daily.

* According to Jaccoud, the cases of cure recorded are examples of hyperæmia or meningitis.

CHRONIC MYELITIS.

Again it will be necessary to remind the reader that attention is here directed to those inflammatory processes alone which attack the cord in a promiscuous fashion. In other words, no reference will be made to chronic lesions of the special physiological systems; as, for example, primary posterior and lateral sclerosis, and anterior cornual degeneration or progressive muscular atrophy (so-called "system diseases").

MORBID ANATOMY.

As in the case of acute myelitis the lesion will be found to vary very much in situation and extent, so that an accurate diagnosis would include the position and extent of the morbid condition in addition to its nature and intensity. In many cases the morbid changes can only be discovered by aid of the microscope, as the naked eye appearances may be those of a normal cord. In brief, the changes met with in chronic myelitis are as follows:—sclerosis and gray degeneration—cicatricial changes which often begin in the nerve cells and fibres, but may commence in the connective tissue or neuroglia; hypertrophy of the axis cylinders, which are seen on longitudinal section to present a varicose appearance; and softening.

In the majority of cases these conditions are more or less combined; when the process has originated in an acute attack, softening is more abundant, but when the lesion is essentially and *ab initio* a chronic one, it is usually more of the nature of a sclerosis. The secondary ascending and descending degenerations are very frequently the result of chronic myelitis.

ETIOLOGY.

Want, over-exertion, alcoholic and sexual excesses, syphilis, hereditary predisposition to nervous affections, with middle age and the male sex, are the most common predisposing causes; and the following operate as exciting causes of the affection:—exposure to cold and wet, injuries to the spine, disease of the bodies of the vertebræ and other factors in slow compression of the cord, syphilis, the specific fevers, and irritation from peripheral organs — so-called reflex paralysis. Lastly, chronic myelitis occurs frequently as a sequel of the acute form of the disease.

SYMPTOMS.

The great majority of the cases of paraplegia depend upon chronic myelitis. The symptoms develop slowly, and usually subjective sensations, or paræsthesiæ—*e.g.* “needles and pins” and “numb-

ness"—first invite the attention of the patient to his spinal weakness. Subsequently the limbs are felt to be heavy, and walking induces fatigue. Other sensory disturbances develop, of the nature of the girdle sensation, more or less backache, anæsthesia, &c. At times slight jerking of the legs is observed, but symptoms of motor irritation are rarely prominent. The feeling of heaviness in the limbs and fatigue on exertion gradually increases, or, as obtains in some cases, exercise may appear to relieve the feeling of weakness and heaviness experienced on first attempting to walk. By degrees the motor weakness increases, evacuation of the bladder becomes difficult, and constipation is complained of. It should not be overlooked that occasionally instead of difficulty of micturition and constipation the sphincters may be relaxed from the beginning. As the disease advances the paralysis, motor and sensory, becomes more pronounced, and the case may either assume the proportions of complete paraplegia, or stop short at a condition of partial paralysis, and show a disposition to remain in the same state for a long time. Bed-sores and cystitis are by no means so frequent as in acute myelitis; nevertheless, as the paralysis becomes more pronounced, they occasionally develop in the chronic affection as well. The behaviour of the reflexes depends essentially upon the posi-

tion and distribution of the lesion. When the focus of myelitis is situated above the lumbar enlargement the lower skin reflexes (plantar and cremasteric) are generally increased; so may the abdominal and epigastric, but this is contingent upon the height of the lesion. The knee-jerk is also commonly increased in cervico-dorsal and lumbo-dorsal lesions. When the lumbar enlargement is extensively diseased the reflexes are abolished. The amount of wasting and the electrical reaction of the paralysed muscles also depend upon the position and extent of the lesion. Some chronic cases suddenly assume the features of an acute attack, and terminate in rapidly ascending softening of the cord.

The following are the principal varieties of chronic myelitis:—

1. *Chronic Central Myelitis*, or inflammation of the central gray matter, which stands in a generic relation to Hallopeau's paralysis, or *perispinal myelitis*. This latter affection is characterised by a somewhat rapidly developed atrophy and paralysis of the thenar and hypothenar muscles of the hands, the extensor and flexor muscles of the fingers, and interossei. Other muscles of the forearm and arm subsequently become affected, and the lesion may either ascend or descend the cord, but the process often remains stationary for a few months.

Under this heading is also included the *sub-acute general spinal paralysis* of Duchenne, which is essentially a chronic atrophic paralysis. In this form of myelitis the clinical features resemble those of Hallopeau's paralysis very closely, except that the paralysis generally begins in the lower extremities. According to Ross* it is a chronic lesion of the "fundamental ganglionic cells" of the anterior horns (trophic centres); whilst periependymal paralysis is, in the opinion of the same writer, a lesion of the same column of cells only slower in its development. These two affections are marked by a progressive atrophic paralysis, with early loss of electrical reaction, without bed-sores or sphincter paralysis, which may pursue an ascending or a descending course, and in which, whilst there may be a certain degree of analgesia, anæsthesia is not a feature.

2. *Chronic Transverse Myelitis*, in which almost the entire transverse section is affected. Although slow compression of the cord might well be included under this heading, I prefer, on account of the extreme importance of the subject, to consider it separately. Transverse myelitis of the cervical, dorsal, or dorso-lumbar regions of the cord is very commonly attended with secondary descending and ascending degenerations, and therefore it is usual to meet with rigidity of the

* "Diseases of the Nervous System," vol. ii., p. 300.

limbs and increased tendon-jerk phenomena in addition to the backache, girdle pain, subjective sensations, cramps in the legs, difficulty in micturition, and motor and sensory paralysis. In fact, chronic transverse myelitis, including *compression myelitis* as well as primary inflammation, is the commonest cause of *spastic paraplegia*.

3. *Chronic Progressive Myelitis*,—an affection which is characterised by muscular weakness and paralysis commencing in the lower extremities, and slowly ascending, so as, subsequently, to involve the upper; and in which the muscles eventually waste, whilst the electrical irritability and reflexes disappear. Backache, subjective sensations, and anæsthesia are generally pronounced, and bed-sores and paralysis of the sphincters usually terminate the case.

The remaining forms of chronic myelitis, chronic disseminated myelitis or multiple cerebro-spinal sclerosis, and chronic bulbar myelitis or bulbar paralysis, demand a special notice.

PROGNOSIS.

The prognosis of chronic myelitis is essentially unfavourable; but some cases, after a protracted course, recover up to a certain point, though complete recovery is very rare. Cases of myelitis due to slow compression and syphilis are the most favourable.

TREATMENT.

The essentials in the treatment are:—counter irritation to the spine, especially the actual cautery; ergot and belladonna, either by the mouth, or in the form of ergotin and atropia administered hypodermically; cold water cure, as advocated by Erb*; electricity—the galvanic current to the spine and the interrupted to paralysed muscles; and “medical rubbing,” which certainly deserves a trial especially in cases of atrophy from disuse†; and nerve tonics, such as tribasic phosphate of silver ($\frac{1}{4}$ — $\frac{1}{2}$ grain doses), phosphorus, iron, quinine, strychnine (which is only admissible when no symptoms of irritation are present), &c. In some cases Fellowes’ syrup of the hypophosphites is very serviceable.

* Ziemssen’s “Cyclopædia,” English translation, vol. xiii., p. 461.

† See paper by Dr. Fletcher Little, *British Medical Journal*, vol. ii., p. 351, 1882.

CHAPTER XXXI.

SLOW COMPRESSION OF THE CORD

(Compression Myelitis).

MORBID ANATOMY AND ETIOLOGY.

CARIES of the vertebræ (Pott's disease), cancer of the vertebræ, and tumours of the membranes, are the commonest causes of slow compression of the cord. Of these three morbid states the first is *par excellence* the most frequent cause of the affection under consideration.

Pott's disease induces compression of the cord in the great majority of the cases by setting up an external pachymeningitis,* in which the caseous or scrofulous deposit that appears on the surface of the dura mater compresses the adjacent nervous structures, cord and nerves.

Cancer of the vertebræ, seldom primary, is a much rarer cause of compression; yet it is one that operates in a considerable number of cases. In this condition the nerve trunks are often compressed, whilst the cord itself may escape.

Tumours which exert pressure on the cord are either intra-medullary, or grow from the meninges.

* See p. 257.

The latter, the most common, are very seldom malignant, and generally spring from the inner surface of the dura mater.*

Of the tumours of the substance of the cord, the solitary tubercle is the commonest; gliomata and gummata are also occasionally observed, but appear to be very rare.

Internal pachymeningitis when attended by an extensive hæmatoma will induce symptoms of compression.

It may be accepted without doubt that very few spinal cords subjected to compression by any of the ways indicated above escape inflammatory changes. Admitting the fact that cases do occasionally arise in which the symptoms are due to simple compression alone, I desire to emphasise the statement that sooner or later the cord inflames in compression cases, and I deem it the more necessary to do so as it seems to be very generally held, especially as a *surgical* opinion, that morbid changes in the cord are the exception in Pott's disease, rather than the rule. In general, the medullary changes are of the nature of a transverse interstitial myelitis, "recalling the characters of sclerosis, and accompanied by a more or less complete destruction of the nerve-tubes.†" The

* Sarcomata and psammoma are met with here.

† Charcot, "Diseases of Nervous System," 2nd series, p. 78 of the New Sydenham Soc. Trans.

antero-lateral portion of the cord is most frequently exposed to compression; and the dorsal and dorso-lumbar regions are the situations commonly attacked, but the cervico-brachial is also at times the seat of the lesion. Secondary degenerations are exceedingly common in compression myelitis.

SYMPTOMS.

The symptoms generally commence with subjective sensations ("needles and pins," &c.) in the limbs which subsequently become paralysed; but in some cases motor disorders are first complained of, such as, gradually increasing paralysis with flaccidity of the limbs, and subsequently irritative phenomena—cramps, &c. Later on rigidity makes its appearance; at first there is forced extension, but afterwards contraction of the flexors, and consequently flexion.

The skin reflexes and tendon-jerk phenomena are commonly very pronounced.

Neither anæsthesia nor analgesia find a place among the early symptoms of compression myelitis; indeed it is a notable fact, and one of great diagnostic value, that unless the nerve trunks are implicated there is generally an entire absence of marked anæsthesia until late in the history of the case. At the same time it must be remarked that on account of the block in the cord there is often a delay in the transmission of impressions; and,

further, there may be a difficulty experienced in referring the impression to the spot stimulated, and the sensation may persist after the stimulus has been withdrawn (*persistent after sensation*).

Although sensory disturbances are not a feature of compression myelitis proper, nevertheless they occupy so prominent a place in the clinical picture of the case, in the form of characteristic *pains* (accessory symptoms depending upon the cause of the compression), as to make them of paramount importance. These *pains* may be localised in the back at the seat of compression and *neuralgic-like*, or radiating in the limbs and trunk; the former are due to irritation of the membranes, and the latter to irritation of the posterior roots of the spinal nerves. They are generally complained of for some time before the symptoms of myelitis make their appearance.

The muscles may maintain their normal plumpness and electrical reaction for a very considerable time; but, unless the cause of compression be arrested, atrophy will certainly ensue, either from disuse, or from an extension of the myelitic process to the anterior cornual cells. In the former case there will be a diminished reaction to both forms of current, and in the latter, after a short period marked by the *reaction of degeneration*, the strongest current will fail to elicit any response. As a rule, the patient complains of a

difficulty in emptying the bladder, but only when the lesion is very low down in the cord is there paralysis of the sphincter.

DIAGNOSIS.

Whatever the cause may be, the symptoms of compression proper are always the same, and when taken along with the *pains* and other intrinsic signs, are truly characteristic. Thus, the gradually increasing motor paralysis, rigidity, pain in the back and shooting pains, without anæsthesia, along with a prominent vertebral spine, leaves no room whatever for doubt.

In Pott's disease the spinal deformity is more angular than in cancer, and the pain is less pronounced. The pains of the latter affection are often of the girdle description, and are paroxysmal, being worse at night. When the lesion is in the cervical region the arms may be paralysed, and the legs escape;* but in severe compression of this portion of the cord both arms and legs are affected. It is important to distinguish cervical paraplegia due to cord compression from that due to compression of the nerve trunks. In the latter case the loss of motor power is accompanied directly by acute neuralgic-like pains, and is followed

* According to Brown-Séquard, this is to be accounted for on the supposition that the motor conductors for the upper extremities lie more superficial than those for the lower.

sooner or later by anæsthesia, rapid atrophy of the muscles, and loss of faradic contractility and reflex action in the paralysed limbs.

In cervical paraplegia there are often changes observed in the pupils, which may be dilated or contracted for weeks at a time. Cough, dyspncea, vomiting, dysphagia, and slow pulse are also occasionally met with when the lesion is high up.

When the lumbar enlargement is seriously compressed the limbs remain flaccid, and the sphincters are paralysed, whilst reflex action is abolished.

The symptoms of compression of the cauda equina vary according to the position and extent of the lesion; but, as a rule, there are marked neuralgic pains, motor paralysis, atrophy, and anæsthesia along with rapidly developed bed-sores. In such a case the bladder and rectum often remain intact.

PROGNOSIS AND TREATMENT.

To Charcot we are indebted in a very great measure for our knowledge respecting the curability of compression myelitis due to Pott's disease. He has shown that recovery has taken place even after complete paraplegia, and that when the cord at the compressed part was "no bigger than the barrel of a goose-quill, and in section corresponded to about the third of the section of a normal cord examined in the same region. It was very firm in consistence, and gray in colour; in one word,

the cord presented all the appearances of most advanced sclerosis."* I have no hesitation in saying, from my own experience, that no cases of organic cord lesion are so amenable to treatment as those of compression myelitis from Pott's disease. Of course the prognosis in cases the result of tumours, whose nature it is to advance, and in cancer, is very unfavourable.

The treatment consists in the application of the actual cautery to the affected region of the spine, the administration of ergot and belladonna as long as any signs of irritation last, and subsequently of cod-liver oil, iron, phosphate of silver, &c. The patient should be kept in bed for a few weeks, whilst the cautery is applied from time

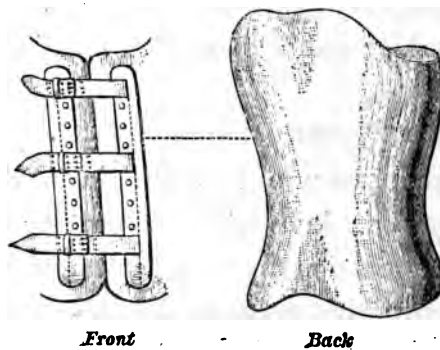


FIG. 41.—*Cocking's Poroplastic Felt Appliance for Spinal Curvature.*

to time, and then may be allowed to get up, encased in some spinal support, such as a poroplastic jacket.

* *Op. cit.*, p. 80.

CHAPTER XXXII.

MULTIPLE CEREBRO-SPINAL SCLEROSIS.

MORBID ANATOMY.

DISSEMINATED sclerosis, or multiple sclerosis of the cord and brain, is seen in the post-mortem room in the form of numerous nodules or sclerosed patches which are scattered throughout the



FIG. 42.—Section of the Brain through the upper part of the Corpus Callosum from a typical case of Disseminated Cerebro-spinal Sclerosis. (For the specimens from which this and the next fig. were taken I am indebted to Dr. Peart, of North Shields.)

cerebrum, cerebellum, pons, medulla, and cord. At times the cord and brain are affected separately, but this is rarely the case. The patches of sclerosis vary in size in the brain from a millet-seed to a hazel-nut; but on becoming confluent, they may attain to a much larger size. In the cerebrum they usually occupy the white

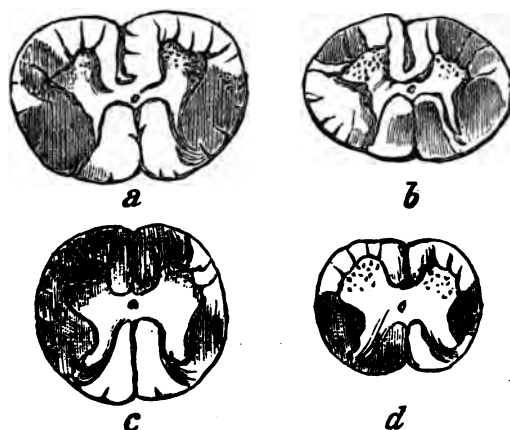


FIG. 43.—Sections of the Cord at different levels, showing the erratic distribution of the lesion.—*a*, middle of the cervical enlargement; *b*, lower part of the same; *c*, middle of the lumbar enlargement; *d*, lower part of the same.

matter, and the cortical substance is comparatively seldom attacked. In the cord there is great variety in their distribution; indeed, each successive transverse section may reveal a new nodule in a new situation, ranging in size from a hemp-seed to a small bean (*see fig. 43*). They are grayish-yellow, or salmon-coloured on

section, and present to the touch a somewhat firm surface. The cranial nerves and anterior and posterior spinal roots are sometimes attacked by similar patches of sclerosis. The appearances of the morbid structure on microscopical examination are those of chronic interstitial myelitis; thus the neuroglia network is thickened and rendered more dense, whilst the medullary sheaths of the nerve-fibres are destroyed. For a considerable time the axis-cylinders remain intact, but ultimately the patch of sclerosis is reduced to a mass of wavy fibrillated connective tissue from which all trace of nerve structure has disappeared. It is a remarkable fact that a cord affected with disseminated sclerosis usually escapes secondary degeneration for a long time. In advanced cases the nuclei in the medulla and the cells in the anterior and posterior horns undergo degeneration, and a large number of amyloid corpuscles are found scattered through the cord.

ETIOLOGY.

Multiple cerebro-spinal sclerosis seems to occur most frequently between the ages of twenty and thirty,* and it has very seldom been known to make its appearance after forty-five. In some

* Cases are met with from time to time in children. I myself have seen a case in a girl of three years.

recorded instances the disease was evidently hereditary, but such is not very commonly the case. Exposure to cold and wet, excessive mental and physical exertion, and syphilis are reputed amongst the most frequent exciting causes of the affection. The two sexes are about equally attacked.

SYMPTOMS.

Charcot has divided disseminated sclerosis into the cerebro-spinal, the cerebral, and the spinal forms.

Cerebro-spinal Sclerosis.—This form is by far the most frequent and characteristic. The initial manifestations may be referred either to the brain or to the cord. In the former case the early symptoms are headache, vertigo, paralysis of the cranial nerves, &c.; in the latter, they consist of paræsthesiæ, gradual loss of power in the legs, unsteady gait, and shooting pains. As a rule, the weakness distinctly commences in one leg, and subsequently extends to the other, and eventually to the arms. Occasionally the onset is sudden, when a fit, soon followed by hesitating, scanning speech, are the first symptoms.

The most characteristic feature of the disease is a *peculiar volitional tremor*, which makes its appearance when any voluntary effort is made. This tremor affects the arms, legs, and head, and can easily be distinguished from the tremor of paralysis agitans on the one hand and from

chorea on the other by the fact that the shaking appears almost exclusively during voluntary movements, and disappears during repose; and further, the excursions or oscillations are more extensive than those of shaking palsy, but not so extensive as, and much less erratic than, the choreic movements.

The tremor of disseminated sclerosis is well seen when a patient attempts to seize and raise to his mouth a tumbler of water. This almost pathognomonic phenomenon is probably only present when the cerebral centres (medulla and pons) become affected. The gait, which is usually of the spastic variety, is very suggestive, especially when the tremor is well marked.

As the paralysis becomes more pronounced, muscular rigidity sets in, and eventually the flexors contract. Anæsthesia is not often an early feature, but it may develop, varying in degree from time to time, often limited in its distribution, and accompanied with an impairment of the muscular sense.

The sight is often affected, and the optic discs may show marked atrophic changes. Nystagmus or oscillation of the eye-balls, scanning or syllabic speech, and tremulous tongue, are all symptoms which make their appearance as the disease advances. The speech is peculiarly characteristic; it is slow and hesitating, and the patient drawls out and lingers over each syllable.

The plantar reflex is seldom much altered, but the

303 *Disseminated Cerebro-spinal Sclerosis.*

cases vary very considerably in this respect; the knee-jerk is usually exaggerated, and ankle-clonus is frequently present. As the affection develops locomotion becomes impossible, and eventually the erect position cannot be maintained. In some cases this inability to stand or walk is due to the rigidity and tremor of the legs, but in others it depends absolutely upon paralysis. Until very late in the history of the case the paralysed muscles are free from wasting, and their electrical condition continues normal. The vesical and rectal functions remain unaltered, or, are seldom interfered with for a long time, constipation being commonly the only inconvenience.

Psychical disturbances make their appearance sooner or later in nearly all the cases. They consist of emotional excitability, loss of memory, and general mental obtuseness, which occasionally culminate in a state of utter dementia.

During all this time the volitional tremor* and scanning speech continue as prominent features. Ultimately the general nutrition becomes impaired, when the limbs atrophy and contract, and bed-sores form; and towards the end more pronounced bulbar symptoms develop in the shape of difficulty in swallowing, and paralysis of the respiratory muscles.

In the cerebral variety of disseminated sclerosis

* In the later stages of the disease the tremor shows a tendency to become constant, but it is always worse on exertion.

the mental phenomena are predominant; and, further, the tremor and syllabic speech not only precede, but greatly exceed the paralysis. As already stated, it is rare to meet with cases in which the sclerosed nodules are confined to the brain.

In disseminated inflammation of the spinal cord or multiple spinal sclerosis, which is also a rare disease, the cerebral symptoms, *e.g.*, volitional tremor, nystagmus, psychical manifestations, disturbances of speech, &c., are absent. As a rule, such cases unite in a suggestive way a good number of the features of a lateral sclerosis to those of a case of locomotor ataxy. The gait is uncertain and somewhat staggering, shooting pains are generally present, and co-ordination is defective; whilst there is a certain amount of rigidity, motor paralysis, and increased knee-jerk.

DIAGNOSIS.

Cases of multiple cerebro-spinal sclerosis are seldom difficult to recognise: the union of spinal with cerebral symptoms, the combination of a slowly developed paraplegia with volitional tremor, scanning speech, and nystagmus, constitute a clinical picture which cannot well be mistaken.

The diseases most liable to be mistaken for disseminated sclerosis are paralysis agitans, cerebellar disease, and locomotor ataxy.

Shaking palsy, or paralysis agitans, is dis-

tinguished by the absence of head symptoms, the late appearance of the paralysis, *i.e.*, after the tremor has lasted for some time, and by the fact that the tremor is entirely independent of voluntary movements.

From disease of the cerebellum cerebro-spinal sclerosis is distinguished by the absence of optic neuritis,* vomiting, and paroxysmal headache. In both cases there may be volitional tremor, syllabic speech, and nystagmus, but these symptoms are comparatively rare in cerebellar tumour; whilst in the latter affection inco-ordination of station and reeling gait are predominant.

Multiple sclerosis of the brain and cord is known from locomotor ataxy by the characteristic speech, tremor, and psychical disturbances which are present in the former and absent in the latter; and, further, the sensory disorders, analgesia, retarded sensibility, and lightning pains, are much more pronounced in locomotor ataxy.

PROGNOSIS.

The prognosis is always unfavourable; but in many cases it happens that the course of the disease is interrupted by periods of temporary improvement. The average duration is from five

* It is generally cerebellar tumours which are confounded with multiple sclerosis, and optic neuritis is one of their most prominent features.

to ten years; however, though some cases have lasted much longer, others have been known to terminate fatally in from one to two years.*

TREATMENT.

Hammond recommends the application of the continuous current to the brain and spinal cord, and the administration of chloride of barium in doses of the eighth of a grain in solution in water, and tincture of hyoscyamus in doses of from one to two teaspoonfuls three times daily. According to Ross,† “the most promising treatment appears to be the persistent application of the galvanic current to the spine, hydropathy, nitrate of silver, phosphorus, cod-liver oil, and nourishing, but unstimulating diet.” Whatever plan is adopted, it must be recollected that the treatment is really only palliative, at all events in pronounced cases.

* In a typical case at present under observation the symptoms are of fully twenty years' standing.

† “Diseases of the Nervous System,” vol. ii., p. 813.

CHAPTER XXXIII.

LANDRY'S PARALYSIS

(Acute Ascending Paralysis).

By acute ascending paralysis is meant a form of acute motor paralysis, described by Landry in 1859,* which generally attacks the lower extremities first, and spreads rapidly upwards, so as to engage the cervical enlargement and medulla.

MORBID ANATOMY.

With one or two exceptions, no morbid changes have been discovered in the spinal cord, brain, and peripheral nerves in patients who have succumbed to this disease. Déjerine and Gœtz found changes in the anterior roots; but Erb concludes that, inasmuch as the examination was not thorough, the observations are of doubtful import. Ross† found marked changes in the central gray column and central group of cells, but he appears to doubt the accuracy of the ante-mortem diagnosis. Some writers regard this affection as

* Although Landry was the first to call attention to this disease as a special affection, cases had been met with, and recorded previously; and it is supposed that Cuvier died of it in 1832.

† "Diseases of the Nervous System," vol. ii., p. 270.

analogous to tetanus, or, as a form of blood-poisoning.

ETIOLOGY.

To say that the causes of acute ascending paralysis as described by Landry are imperfectly known is barely stating the truth; for the fact is that very little, if anything, is understood respecting the causation of the disease; indeed, our information will only warrant the general statement that the affection has been noted more frequently in men than in women, and occurs after twenty years of age, whilst exposure to cold, syphilis, acute infectious diseases, and malarial intoxication, have been regarded as causes.

SYMPTOMS.

In most of the recorded cases a premonitory stage marked by febrile symptoms and obscure pains has been observed. This stage has been known to last several weeks, but it rarely extends beyond a few days. The disease then reveals itself by motor paralysis of the lower extremities, which rapidly advances, so that very soon standing and walking become impossible. The paralysis appears first in the muscles engaged in the movements of the feet, then in those of the legs and thighs, and very soon the muscles of the trunk and upper extremities fall victims in their turn to the progress

of the affection. The paralysed muscles are observed to be perfectly flaccid, and, though somewhat wasted, do not undergo marked atrophy; whilst their electrical reaction remains normal. The skin reflexes, though at first unaltered, soon become diminished, and the tendon-jerk phenomenon disappears. Very little, if any, anæsthesia is present; there are no bed-sores, and there is no affection of the bladder and rectum. As the disease pursues its course upwards there is stiffness and paralysis of the muscles of the neck, the functions of the important centres in the medulla are interfered with, and the patient usually dies of asphyxia in from eight to twelve days, without developing cerebral symptoms.

DIAGNOSIS.

From acute anterior polio-myelitis Landry's paralysis may be distinguished, for it pursues a progressive course, attacking muscle after muscle; and, further, in the former affection the muscles waste rapidly, and the faradic excitability is early lost. In acute central myelitis—a disease which may readily be confounded with acute ascending paralysis—there are marked disturbances of sensibility, the sphincters suffer, and bed-sores make their appearance.

There is a form of progressive paralysis that occurs in intemperate women which may be mis-

taken for acute ascending paralysis. It shows itself, after a preliminary stage marked by shooting pains in the legs, with, perhaps, intermittent attacks of motor weakness, by a more or less general motor paralysis, especially marked in the legs, and by marked hyperæsthesia of the skin* and muscles; whilst the latter waste, and lose their electrical excitability. The disease generally advances upwards, and the case may terminate with coma and other symptoms indicative of cerebral implication, in a few weeks.

PROGNOSIS.

The prognosis in Landry's paralysis is always most grave. Some cases have proved fatal in two or three days; as a rule, death occurs within a fortnight from the beginning of the paralysis. Some have been known to recover, but when the affection rapidly ascends the cord and attacks the medulla the case is hopeless.

The plan of treatment recommended in acute myelitis may be adopted in acute ascending paralysis.

* Some of these cases present analgesia of the skin, and appear to run a course very similar to chronic atrophic spinal paralysis (*see* p. 316).

CHAPTER XXXIV.

 LESIONS CONFINED TO THE ANTERIOR CORNUA.

CHRONIC ATROPHIC SPINAL PARALYSIS*

(*Sub-acute Inflammation of the Anterior Cornua*).

THIS is a rare, comparatively chronic, affection that generally begins in the legs and shows itself by motor paralysis and muscular atrophy, which gradually make their way upwards.

MORBID ANATOMY.

In the recorded post mortems of this disease the large cells of the anterior cornua have been found to be markedly affected. In one case recorded by Ross, "the ganglion cells of the anterior gray horns had almost completely disappeared throughout the entire length of the spinal cord."†

ETIOLOGY.

Blows on the back, exposure to cold, plumbism, and *alcoholic excesses* are said, occasionally, to

* "Paralyse Générale Spinale Antérieure Subaiguë (Duchenne)."

† "Diseases of the Nervous System," vol. ii., p. 141.

stand in a causal relation to this disease; but it must be admitted that, with the exception of *alcohol*, its etiology is very imperfectly understood.

SYMPTOMS.

The earliest phenomena are usually a sense of fatigue after exercise, with obscure pains, and, it may be, subjective sensory disturbances. Then motor weakness makes its appearance, and gradually increases until complete motor paralysis is established. The affection, as already stated, first attacks the legs. The paralysed muscles are painful, flaccid, and soft, and no resistance is offered to passive movements of the limbs. Fibrillation may be present, and rapid atrophy ensues, but the paralysis in every case precedes the wasting; and early in the case the "reaction of degeneration" is observed on the application of the electrical tests. Sensation is not much altered; bed-sores do not develop; and the rectal and vesical sphincters are not often interfered with. The paralysis gradually extends to the upper extremities, where certain groups of muscles are selected at first, at times the extensors and anon the flexors, but sooner or later the whole limb may become paralysed and atrophied. The muscles of the trunk also become involved, and coughing, sneezing, and defecation are carried on with difficulty. All reflex action is abolished

in the paralysed limbs, and soon all electrical excitability vanishes.

The onward progress of the disease is often temporarily arrested, and indeed, recovery may take place; but should the lesion continue to advance the medulla is invaded, and death results from respiratory paralysis.

PROGNOSIS.

Generally speaking, as in the case of infantile paralysis, the prognosis is favourable as regards life; in some cases complete recovery takes place, but probably in the majority the cure is only partial.

TREATMENT.

The spinal ice-bag and other antiphlogistic measures should be used at first; and subsequently the continuous current may be applied to the spine and the interrupted to the muscles, with the administration of tonic remedies.

POLIOMYELITIS ANTERIOR ACUTA.

Acute inflammation limited to the anterior gray horns occurs generally in children, but occasionally the same region is similarly affected in adults.

POLIOMYELITIS ANTERIOR ACUTA INFANTUM

(Infantile Paralysis).

Infantile paralysis is the result of an acute inflammation of the anterior horns, which commonly occurs in the lumbar or cervical enlargement, or both, but which may also involve the dorsal portion of the cord. The most important structures affected are the ganglionic cells; but whether the inflammation commences in the cells themselves or in the neuroglia is not quite determined, though all seem to agree that it is both parenchymatous and interstitial. Under the microscope the large multipolar cells with their axis-cylinder processes are seen to be more or less extensively destroyed; whilst there is a proliferation of nuclei, and dilatation of blood vessels. At a later stage the affected ganglionic cells entirely disappear (*see* fig. 44), and the horns become shrivelled and atrophied.

Occasionally the lesion extends at certain points beyond the limits assigned to it in the foregoing description, so as to affect the posterior horns or the antero-lateral columns, but these additional changes are essentially secondary.

The anterior spinal roots and the peripheral nerves also undergo atrophic changes.

The paralysed muscles atrophy; and the degenerated fibres, when examined microscopically,

are seen to be granular and infiltrated with oil globules, and to have lost their normal striation. As a rule healthy as well as degenerated fibres

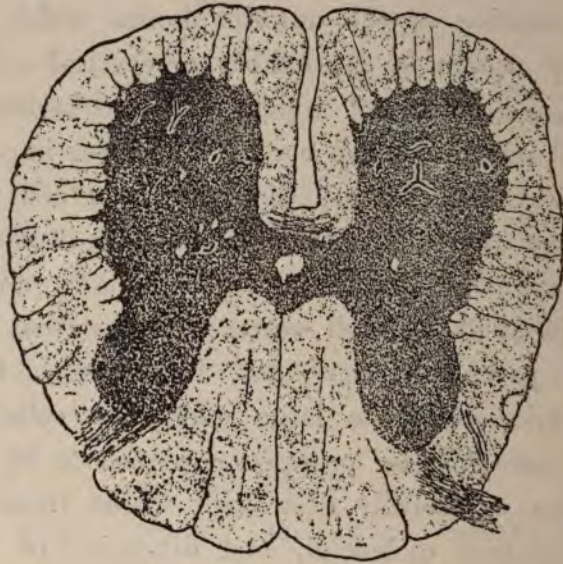


FIG. 44 (after BRAMWELL).—*Transverse section of the Cord in the lumbar region, from a case of infantile paralysis—the majority of the anterior cornual cells have disappeared.*

are observed in the same muscle. In old-standing cases the degenerated muscles are composed almost entirely of connective tissue and fat; and the tendons are wasted and transformed into thin bands.

ETIOLOGY.

Very little that is reliable can be advanced under this head. The majority of the cases occur

between six months and four years, though the affection may make its appearance at an earlier period, and, as we have seen, cases of acute anterior poliomyelitis are occasionally met with in the adult.

From my experience at the Children's Hospital, Newcastle-on-Tyne, I am inclined to attach great importance to the process of dentition as a factor in the production of the disease, though some writers make light of it. Exposure to cold, scarlatina, and other febrile diseases are set down as probable causes of infantile paralysis.

SYMPTOMS.

In most instances the first departure from health is a feverish attack, which may be attended by convulsions but which much more commonly differs in no way from an ordinary attack of "teething" without eclampsia. At times no such history of initial febrile disturbance can be elicited, especially in very limited lesions. The febrile symptoms usually last a few hours before the onset of the paralysis, which apparently takes place suddenly, or, to speak more correctly, develops in a few hours, and is only detected after its complete development. Occasionally several attacks occur one after the other, and may be separated by an interval marked by an abatement of the febrile symptoms. Sometimes

only a single group of muscles is paralysed, at others an entire limb suffers, whilst both legs may be affected, or a leg and an arm on the same side or on opposite sides; or else, all four extremities may be involved, and not rarely the limbs escape and the muscles of the back are attacked. The paralysed limb is flaccid, and hangs down helplessly. At first it is hot to the touch, and may be hyperæsthetic.

Very often some of the paralysed muscles recover in a few days, while the remainder continue permanently paralysed. All the muscles to which motor power is not restored within a week or two undergo a rapid and progressive atrophy, which makes its appearance in from seven to fourteen days, and becomes pronounced within two or three months from the commencement. Beyond the initial hyperæsthesia just referred to, sensibility remains unaffected. Reflex action is lost in the paralysed muscles, and the knee-jerk is abolished. The bladder and rectal functions continue to be performed in a normal fashion, and bed-sores do not form.

The electrical reactions are extremely important and amply repay attention. In testing the muscles and nerves both forms of current must be employed. Muscles which are affected by permanent paralysis lose their faradic response in about ten days or a fortnight, but those about

to recover are still irritable. The nerves permanently lose both their faradic and galvanic excitability early in the case, but the muscles behave differently; thus, by the end of the first week the muscles show a marked diminution of response to both forms of current, and whilst, as the case progresses, the faradic excitability continues to lessen until it is soon completely abolished, *the galvanic excitability* (by the second week) *begins to rise, and soon becomes exalted beyond the normal.* Further, the character of the response is altered; for, instead of the quick contraction on the *make* and *break*, which is a feature of health, the response is sluggish and shows a tendency to sustain itself during the whole time the current is passing. This constitutes the *reaction of degeneration*, a phenomenon which lasts in infantile paralysis for upwards of six or eight weeks, after which there is a decided diminution in galvano-muscular excitability; and subsequently the paralysed muscles refuse to contract to the strongest galvanic current.

During the late stages of the affection there is an arrest in the growth of the bones and joints, which become exceedingly mobile, so that the limb may be tossed about like a flail; and the flaccid, wasted member shows a decided lowering of temperature. Finally, contractions and deformities occur: the great majority of the deformed

mendicants about our streets owe their distortions to an attack of anterior poliomyelitis in early life.

PROGNOSIS.

A fatal termination is exceedingly rare; but, according to Althaus,* a certain number of the fatal cases of "convulsions" in infancy is to be ascribed to "severe inflammation of the anterior cornua of the cord."

So far as complete recovery of the paralysed limb is concerned the prognosis is unquestionably unfavourable. The muscles which remain paralysed, and in which the faradic excitability is lost at a period of ten days from the onset, will, in all probability, not recover. Some cases, examples of the so-called "temporary spinal paralysis," recover very rapidly, within a day or two from the onset, but these are rare, and are, in the opinion of the author, usually a phenomenon of "*teething*." In all cases a certain degree of improvement may be looked for, but complete recovery can very seldom be predicted.

TREATMENT.

Should the child be seen during the feverish attack which usually ushers in the paralysis, ergotin may be injected subcutaneously, as re-

* "On Infantile Paralysis," p. 12.

commended by Althaus, op. cit.* Iodide of potassium may be employed along with counter irritation to the spine when the fever has subsided. Subsequently electricity must be used, and that form of current especially which contracts the muscles. Friction with a stimulating liniment, and muscle-rubbing (*massage*) are extremely useful; and finally, cod-liver oil should be given, and the tendency to deformity counteracted by appropriate orthopædic appliances.

POLIOMYELITIS ANTERIOR ACUTA
ADOLESCENTIUM.

Acute inflammation of the anterior horns is a very rare disease in the adult; nevertheless, cases are recorded from time to time. The lesion is precisely the same as in the case of infants. The onset is attended by pain in the back and extremities, with a certain amount of initial fever; and the paralysis is developed within a day or two of the commencement of the symptoms.

As in the case of children, the paralysed muscles rapidly waste, and when tested electrically, show evidences of the *reaction of degeneration*.

* "One-fourth grain for a child from one to two years of age; one-third grain for one from three to five; half a grain for children from five to ten years of age; and a grain for patients upwards of ten."

tion. The limb becomes cold, and a certain degree of contraction results; there is no permanent disturbance of sensibility, nor do bed-sores or vesical trouble enter into the case.

The treatment is the same as in the infantile variety of the affection.

PROGRESSIVE MUSCULAR ATROPHY

(*Wasting Palsy*).

This affection, when manifested in a typical way, is the commonest form of chronic and progressive wasting of the muscles that comes within the range of cord disease. Charcot* gives it the name of "*protopathic spinal progressive atrophy*," in contradistinction to the *deutero-pathic*† varieties of muscular atrophy, signifying thereby that the trophic disturbance is the primary and essential feature of the disease.

MORBID ANATOMY.

The lesion commences in, and, as a rule, is confined to, the anterior cornua; in some cases, after the lapse of a considerable period, the process, which is most probably inflammatory,

* Charcot "On Diseases of the Nervous System."

† By deutero-pathic atrophy is meant lesions in which the trophic centres of the cord are attacked secondarily.

outsteps the limit thus ascribed to it, and attacks the adjacent white and gray matter of the cord; but the initial, the essential, and often the sole morbid change, is found in the anterior horns. The nerve-cells of this region undergo atrophy, and their processes degenerate; further, there is proliferation of the cellular elements of the neuroglia, and the gray horns are often diminished in size. The anterior nerve-roots and the peripheral nerves degenerate in a more or less prominent way; of course, this change is secondary to the atrophy of the cornual cells, and, it should be recollected, is never very pronounced.

The wasted muscles undergo a simple atrophy, but their fibres retain their cross striation. They are pale, and show a tendency to fatty degeneration, which affects, but less apparently than in the case of infantile paralysis, the perimysium as well as the muscle-fibres. The muscular connective tissue is also increased, and, at times, to the naked eye there is little else left to represent the muscle.

ETIOLOGY.

The disease is most frequently met with between the ages of thirty and fifty; and though women are sometimes affected, it is decidedly more frequently developed in the male sex. The

part played by hereditary influences seems to be most important; indeed, most writers are familiar with cases in which several individuals have been attacked in one family.*

As exciting causes, excessive muscular exertion, exposure to cold, intemperance, injuries to the spine and local injuries to muscles, acute diseases, and lead-poisoning, are all believed to exert an influence. However, in the majority of instances, the exciting cause appears to remain a mystery.

SYMPTOMS.

The affection is essentially insidious in its onset. In many cases the disease is sufficiently far advanced to enable an indubitable diagnosis to be made almost as soon as the patient is aware that anything is amiss. Generally speaking, his attention is first called to his complaint by discovering that certain usual movements cannot be performed with customary readiness; this discovery may prompt the statement that the paralysis came on suddenly, as a cartman, at present under my care, informed me, because the difficulty he experienced in holding the reins seemed to him of sudden development.

The paralysis and atrophy, which run in

* Naunyn, of Königsberg, records the history of a family in which the transmission could be traced through five generations.

parallel lines, usually (according to Charcot, "in the immense majority of cases") commence in one of the upper extremities. The interossei muscles, or those of the thenar or hypothenar eminences (opponens pollicis, adductor pollicis, or adductor minimi digiti) are the first to show the change. In some cases the deltoid is the first to be affected, and not rarely the muscles of the chest or back. The lower extremities are only exceptionally invaded first. Duchenne only observed the disease to begin in the legs twice out of 159 cases.

As the muscles atrophy, hollow or flattened regions are left in lieu of the plump contour of the healthy structure; and the actions of the paralysed muscles are lost or limited according to the degree of atrophy. The hollows between the metacarpal bones, the result of wasting of the interossei, together with the separated, semiflexed aspect of the fingers, give the hand a claw-like appearance which gets the name of *main en griffe*. The paralysis and atrophy of the extensors are usually more advanced than those of the flexors; on this account some cases resemble lead paralysis rather closely.

Fibrillary twitchings in the wasting muscles are exceedingly common, and often precede by a considerable time any obvious degree of atrophy. These fibrillations do not belong peculiarly to

progressive muscular atrophy, as some have thought, but are also often observed in purely functional conditions. The faradic contractility diminishes with the degree of atrophy, but the muscles can be excited to contraction by the interrupted current so long as any healthy fibres persist. The same may be said of galvanism. Sensibility is not interfered with, unless the lesion invades other regions of the cord.* The reflexes are at first slightly increased, but soon a diminution supervenes.

The affection is essentially, as its name implies, progressive, and muscle after muscle succumbs to the wasting process. In many cases when the disease has commenced in one hand, and induced a considerable amount of atrophy in the muscles first attacked, the corresponding ones of the other hand next show signs of wasting—in other words, as Bramwell remarks,† “the morbid process extends to the opposite anterior cornua in the segment first affected.”

Later on the lower limits of the anterior horns become involved, and then the legs begin to waste. But though the morbid process commonly descends

* In the opinion of Charcot the arthralgic and muscular pains sometimes complained of in the course of this disease are significant of an extension of the lesion, but I have so frequently met with painful *cramps* as an early symptom that I am inclined to dispute Charcot's conclusion.

† “Diseases of the Spinal Cord,” p. 194.

the cord so as eventually to involve the lower extremities, an event which Charcot tells us is nearly always deferred until late on in the history of the case, yet, in exceptional instances, the disease makes its way directly upwards from the cervical enlargement, and induces a similar atrophy and paralysis in the muscles of respiration and deglutition, a catastrophe which is necessarily decisive and final.

DIAGNOSIS.

It would be almost impossible to mistake infantile paralysis for wasting palsy, as the sudden onset, the early loss of electrical excitability, and the fact that the paralysis distinctly precedes the atrophy in the case of the former render its recognition easy.

Attention to the following points will enable the observer to distinguish chronic atrophic spinal paralysis, or *paralysie générale spinale antérieure subaiguë* of Duchenne from progressive muscular atrophy. In the former affection the paralysis begins, as a rule, in the lower extremities, and is pronounced before the wasting makes its appearance; and, further, the "reaction of degeneration" is observed early in the affected muscles, which subsequently lose their electrical excitability entirely.

Lead palsy (drop-wrist) may be confounded with

progressive muscular atrophy, but the comparatively sudden onset and rapid development, the early loss of electrical contractility, the late appearance of the atrophy, and the limitation of the paralysis to the group of muscles first attacked, in the case of the former, taken along with the *blue line* and other symptoms of plumbism, serve to distinguish them.

PROGNOSIS.

This is certainly a most grave disease, and one that is positively inimical to life. At the same time the morbid process is occasionally arrested permanently, when the patient recovers with a wasted limb; but, in the majority of instances, bulbar symptoms sooner or later supervene, when a fatal termination may early be looked for. The most favourable cases are those caused by excessive muscular exertion, and the least so are distinguished by an hereditary history of the disease. The duration is very various; the average appears to be from eight to ten years. Some patients succumb within two or three years from the commencement; on the other hand, not a few survive twenty.

TREATMENT.

Duchenne has strongly advocated the electrical treatment of the wasting muscles. This line of treatment, to be of any service, should be carried

out efficiently; the continuous current may be applied to the cord whilst the muscles are exercised by the faradic. In addition to the interrupted current, *massage* or muscle-rubbing may be employed to stimulate the muscles, but care must be taken not to over-exert them. The degree of fibrillation may be looked upon as a guide to the success of the treatment, for a diminution in the fibrillary twitchings is indicative of improvement, and *vice versâ*. Cod-liver oil, phosphate and nitrate of silver, strychnia, &c., may be prescribed, but internal remedies are practically useless in progressive muscular atrophy.

CHAPTER XXXV.

PSEUDO-HYPERTROPHIC PARALYSIS.

ALTHOUGH the pathology of the affection is still *sub judice* and its etiology quite undetermined, the fact that it is characterised by a progressive wasting of the muscles and paralysis makes it convenient to consider it in a clinical work along with progressive muscular atrophy, from which it differs by being generally attended by an increase in the bulk of the affected muscles, the age at which the disease makes its appearance, and the order in which the muscles are invaded.

MORBID ANATOMY.

In spite of several attempts to elucidate the nature of pseudo-hypertrophic muscular paralysis by careful examination of the cord, its pathology remains obscure. Charcot and Friedreich regard it as a primary disease of the muscles; Gowers, though he has observed changes in the cord in one case, seems to have adopted a similar view. Ross writes,* "In the spinal cords which I examined, the changes found in the central gray column and

* "Diseases of the Nervous System," vol. ii., p. 210.

anterior gray horns corresponded so closely with those observed in progressive muscular atrophy, that I see no reason to question the essential unity of the two diseases."

Bramwell* has also observed changes in the cord, principally in the lateral portion of the gray matter, but these he looks upon as "congenital malformations, and not the essential lesion in the case." In a cord I myself examined,† from a



FIG. 45.—*Lumbar region of Cord from a case of pseudo-hypertrophic paralysis, showing growth from left lateral column.*

case of pseudo-hypertrophic paralysis, disintegration of the lateral gray network, especially of the left side, and most marked in the lumbar enlargement, was clearly discernible; and, in addition, there was a conspicuous bulging on one part of the left lateral column, due to a cavity containing serum which was surrounded by gray matter, and

* "Diseases of the Spinal Cord," p. 202.

† *Lancet*, Oct. 15, 1881, p. 660. Figs. 45—49 represent the spinal cord referred to.

extended a considerable distance up the cord from the lumbar enlargement. The ganglionic cells



FIG. 46.—Section of the Lumbar Enlargement through the middle of the bulging.

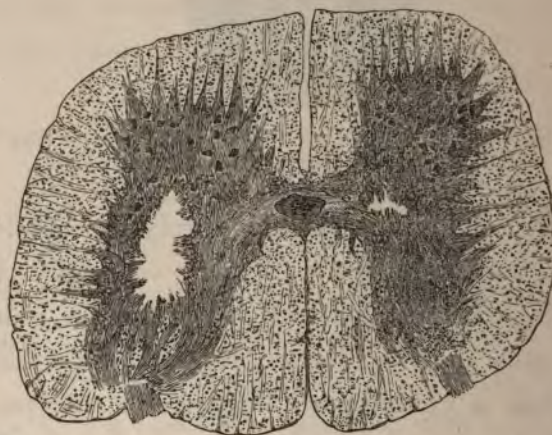


FIG. 47.—Section through the upper part of the bulging.

appeared to be normal. In Gowers' case,* which he examined in conjunction with Lockhart Clarke,

* Gowers' "Pseudo-Hypertrophic Muscular Paralysis," p. 42.

“incipient disintegration in the gray network of the lateral columns adjacent to the gray substance” was noticed. . . “The most extensive lesion



FIG. 48.—Section through the lower portion.

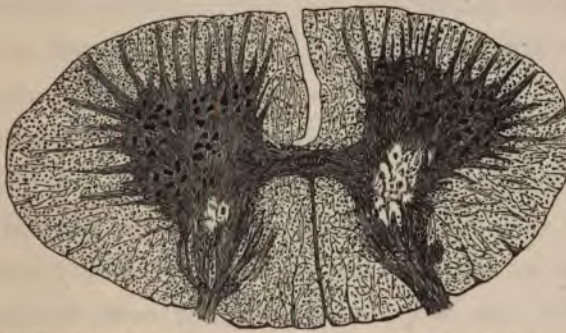


FIG. 49.—Section of the Cervical Enlargement, showing disintegration of the gray matter.

was found in the lowest part of the dorsal region, where in each lateral gray substance was an area of disintegration amounting to an actual cavity outside each posterior vesicular column.”

Whilst reserving judgment upon the part played by the cord in the production of the disease, it is surely suggestive that three cases have shown, in the hands of three separate observers, almost identical changes. At the same time, it must not be forgotten that such competent pathologists as Cohnheim, Charcot, and Meryon have failed to detect anything amiss in the spinal cords which they examined.

At first the only muscular change to be detected is an increase in the areolar tissue which lies between the fasciculi; but soon that which extends between the fibres likewise becomes thickened, thus causing a considerable augmentation of the areolar investment of the muscle. Subsequently there occurs an interstitial and interfibrillary deposit of fat, so that the muscle is much increased in size, and by degrees the muscle-fibres suffer atrophy from compression, the transverse striation being, as a rule, distinct until late on in the disease. Later on, the muscular tissue and adventitious fibrous structure disappear, and eventually nothing remains but adipose tissue, and even from this the fat-cells may in time be absorbed.

ETIOLOGY.

This affection is sometimes, though rarely, met with in the adult; but the great majority of cases occur in early life. Some seem to be congenital;

most frequently, however, the disease manifests itself between five and thirteen years. Boys are more often affected than girls. Out of a total of 220 cases hitherto published, 190 were males and 30 females (Gowers). Typhoid and scarlet fever, and other febrile diseases, have been said to act as exciting causes in some instances.

An hereditary tendency to the disease very frequently exists; and, though the females of a family usually escape the disease itself, the predisposition thereto is transmitted almost exclusively through the female line.

SYMPTOMS.

Awkwardness of gait and feebleness of the lower extremities ordinarily constitute the first reliable signs of this insidious affection. In many cases, however, the hypertrophied calves are remarked before the child makes any attempt to walk; but the true significance of the enlargement is only apparent when repeated failures in this direction have been observed, and that after the age has been passed by a considerable time at which children usually walk. When the onset is deferred until after the child has commenced to walk the gait gradually becomes changed, and the attitude when standing is peculiar. In walking, the legs are kept unnaturally far apart, and the body is moved from side to side resembling

the waddling of a duck. The patient stands with the feet widely separated, and the heels are usually raised slightly off the ground. The back is arched in a curious way, the shoulders and corresponding portion of the vertebral column are carried backwards as a result of a forward inclination of the pelvis and lumbar vertebræ,* with which is carried the dorsal spine. Duchenne made the observation that in extreme cases a vertical line let fall from the middle of the shoulders passes behind the sacrum. As the affection progresses increasing difficulties beset the patient in his attempts to walk and stand, and he is obliged to use his arms, after the manner of a tight-rope walker, to aid him in maintaining his equilibrium. The difficulty in locomotion is intensified by the tendency to the development of talipes equinus, a condition which prevents dorsal flexion of the passive foot as it is being swung forward in walking.

Another, and almost a characteristic feature of the disease is seen in the manner in which the patient attempts to rise from the recumbent to the standing posture. In every instance he will make use of his arms to raise his body; should any articles of furniture be convenient he will drag himself up by them, but in the absence of

* According to Gowers, this lordosis is due to weakness of the extensors of the pelvis on the thighs.

such aid he will "climb up his thighs." He first raises himself on his hands and knees, then upon his hands and feet, then he places one hand above the knee of the corresponding side; this enables him to throw the weight of the body towards the opposite side at the same time that he straightens the knee to which the hand is applied; he may then repeat the same manœuvre with his other hand, when he finds himself in a position to raise himself through the aid of the feeble extensors of the thighs.

The enlargement of the muscles, from which the disease derives its name, is usually an early feature. This pseudo-hypertrophy generally commences in the calf-muscles of one side, but before long the other calf also increases in bulk (*see* fig. 50). Occasionally the enlargement begins in the upper extremities, in which rare instances the deltoids are the first to show the change. After the calf-muscles those of the buttocks are usually the next to suffer, then the lumbar muscles and the thighs become affected, and so on until the majority of the muscles in the body may show signs of the disease.* But commonly the characteristic enlargement is limited to a few muscles; thus, whilst the calves and gluteal

* In one case related by Duchenne, with the exception of the pectoral muscles the latissimus dorsi and the sterno-mastoids, all the muscles of the limbs, trunk, and face were invaded.

muscles are enlarged, the others may be markedly atrophied.

The enlarged muscles may attain a great size;



FIG. 50 (after DUCHENNE).—*Pseudo-hypertrophic paralysis, showing enlargement of the calves and buttocks, and also the spinal curvature—the line A let fall from the middle of the shoulders (fig. to the right) passes behind the sacrum.*

they stand out in a most conspicuous manner, and feel hard like those of an athlete. So long as the morbid process is advancing actively the

temperature of the affected limbs is usually above normal.

In some cases there appears to be no advance made by the disease for a considerable period; indeed, it may be stationary for years. But sooner or later the paralysis and atrophy advance once more, and the case pursues a downward course. The patient may survive for many years in a state of complete paralysis until cut off by some intercurrent disease, most usually of the lungs, or by paralysis of the respiratory muscles.

Sensation is not impaired, and the functions of the bladder and rectum continue to be performed in a normal way.

Lastly, a state of mental enfeeblement has often been noticed to accompany pseudo-hypertrophic paralysis, though in many of the recorded cases the intellectual faculties were normal to the end.

DIAGNOSIS.

Attention to the features referred to will enable the observer to draw a distinction between the affection under consideration and progressive muscular atrophy occurring in infants. In the latter disease, some of the facial muscles show an early change, and subsequently the atrophy extends to the upper extremities, and later still to the muscles of the legs. Occasionally true

hypertrophy of the muscles has been mistaken for the pseudo-hypertrophic enlargement, but the history of the case, the absence of paralysis, and in all cases a microscopical examination of a portion of the muscle removed by the "harpoon" will prevent a mistake.

PROGNOSIS.

In some rare instances the morbid process has been arrested, and the cases have resulted in recovery. But when the disease has established itself in a pronounced way the prognosis is practically hopeless. It has generally been held that the disease runs the most rapid course in very young children.

TREATMENT.

Duchenne asserts that he succeeded in curing two cases in their incipient stages by means of the faradic current. Benedict has also had some slight success with electricity by applying the continuous current to the spine, and the interrupted current to the muscles. Muscle-rubbing may be employed to aid the faradic current in maintaining the nutrition of the muscles. Internal remedies are of very little service.

CHAPTER XXXVI.

LOCOMOTOR ATAXIA

(*Tabes Dorsalis—Sclerosis of the Posterior Columns*).

STRICTLY speaking, locomotor ataxia and sclerosis of the posterior columns are not synonymous titles, though employed as such; the former is descriptive of a leading clinical feature of the disease, and the latter of its morbid anatomy; besides, while the former argues the presence of the latter, yet tracts of the posterior columns may be sclerosed without the characteristic symptoms of locomotor ataxia. In other words, the lesion with which the symptoms are connected is confined to a definite and limited portion of the posterior columns.

ETIOLOGY.

Hereditary influences are said to have an important bearing on locomotor ataxia; occasionally several members of one family are affected with the disorder, but my own experience has led me to the conclusion that heredity has comparatively little to do with its production. The disease is one of early adult and middle life, being

most prone to develop between the ages of 25 and 50. Men are much more frequently attacked than women; out of a total of 149 cases collected by Eulenberg 21 only were females. In the opinion of some writers syphilis underlies the affection in a great many instances. According to Erb about nine-tenths of the typical cases of tabes dorsalis have a syphilitic origin;* but this estimate, arrived at from statistics, is probably much too high. Cold and exposure acting upon a system already predisposed to nervous disorder by want, bodily and mental exertion, or both, in all probability cause most of the cases. Intemperance must also be regarded as a predisposing cause. Sexual excesses and masturbation were formerly held responsible for nearly all the cases of tabes, but at the present time this view is not so much in favour, and the opinion has been advanced, not without reason, that the sexual excess is really the result of the disease rather than its cause, being, in fact, an expression of a state of irritation of the sexual centres in the lumbar enlargement.

MORBID ANATOMY.

We are greatly indebted to Charcot for the knowledge we possess of the precise morbid

* See paper by Prof. Erb before the International Medical Congress held in London, August, 1881.

changes in the cord in this disease. In recent cases the naked eye can detect no alteration, but in old-standing ones the meninges are seen to be thickened and adherent, especially over the posterior columns, and the posterior roots are atrophied, whilst the posterior columns may be shrunk. There is, further, an alteration in the shape of the cord, which is generally flattened from before backwards, as a result of the change in the posterior columns. A section of the fresh cord through the diseased portion reveals a grayish translucent area in the posterior column, which is the cut surface of the sclerosed tract. The consistence of the cord is usually increased. When the cord is examined microscopically the posterior root-zones are found to be diseased in all cases of true locomotor ataxia. The lesion frequently occupies the whole of the posterior columns, *i.e.*, from the posterior median fissure to the outer limits of the radicular fasciculi; but the essential lesion is a sclerous band which occupies the postero-external column, or that portion of the root-zone which borders on the posterior gray horns internally (*see fig. 51*).

The sclerosis is supposed to be of the nature of a chronic inflammation, and apparently commences in the nerve elements, whence it extends to the neuroglia. The columns of Goll, or the median columns, are nearly always affected; but their

implication must assuredly be excluded from all participation in the production of the essential symptoms of the disease. In many advanced specimens the sclerosis has been found to involve the adjacent lateral columns, and posterior and anterior horns; so that the lesion may attack



FIG. 51.—Section of the Lumbar Enlargement of the Cord, from a typical case of locomotor ataxia, showing sclerosis of the postero-external columns.

the crossed pyramidal tracts, the direct cerebellar tracts, and the anterior and posterior cornua, in addition to the essential seat of the disease, viz., the posterior root-zones. The sclerosal atrophy affects the lumbar enlargement most frequently, but it is not unusual to meet with it in the cervical and dorsal regions as well; it may extend into the medulla, and invade some of the bulbar nuclei, but, even in the absence of this special invasion, a degenerated tract can usually be

traced through the medulla as the continuation of the degenerated columns of Goll.

The optic nerve, and occasionally the third, fifth, sixth, auditory, and hypoglossal, may be affected by gray degeneration.

In some cases the joints are markedly affected when the cartilages disappear, and the articular ends of the bones are eroded.

SYMPTOMS.

It is necessary to raise a note of warning at the threshold of a description of the clinical manifestations. Too much importance has hitherto been attached to the *ataxic gait*, and even yet many refuse to believe in the existence of *tabes dorsalis* in the absence of a pronounced inco-ordination in the movements of the lower extremities during locomotion. Whereas, just as in many cases of granular kidney there are, speaking generally, few and but slight symptoms of kidney disease as these were taught by Bright, so it is very common to meet with cases of posterior sclerosis in which muscular inco-ordination is very feebly marked.

The most constant amongst the early symptoms of the affection are *pains* of a characteristic description which are often mistaken for rheumatism, and are variously described by patients. Some compare them to electric shocks, others speak of them as shooting pains, whilst the term "lightning" is very

frequently applied to them. They are, in short, sharp, shooting pains, and are referred to the deeper structures, such as the joints and bones. In connection with this symptom the *gastric crises* (*crises gastriques* of Charcot) deserve special notice. These are severe attacks of gastralgic pain which occur in paroxysms, and are often an early feature. The pains are lancinating, and generally shoot from the lower part of the abdomen to the epigastrium, where they persist for some time and are accompanied by vomiting. The attacks may last for weeks, and during their continuance the patient not unfrequently emaciates considerably. Along with the shooting pains certain paræsthesiæ are usually complained of early, the commonest of which are numbness, needles and pins, the sense of something soft under the feet (furry sensation), and the girdle-sensation. Hyperæsthesia, analgesia (loss of painful impressions), and, in a less pronounced form, anæsthesia (loss of touch), are very often present during the initial stage of the disease, and it is noteworthy that in the majority of cases they occur in localised areas or patches which are surrounded by healthy skin, and, further, manifest a tendency to vary from time to time.

Analgesia is by far the most constant impairment of sensation, and, as the case advances, it may extend so as to affect, in addition to the lower extremities, the trunk, upper extremities,

and even the regions supplied by the fifth pair of nerves.

Besides the more common disturbances of sensibility, the following rarer varieties are often present:—(a) *Retarded sensation*, first described by Cruveilhier. The retardation is most frequently noticed in connection with painful impressions, and may be demonstrated by inserting a pin into the skin, and observing accurately when the sensation of pain is felt; in one case recently under my care twenty-three seconds elapsed before the prick was felt. (b) *Persistent after sensations*, the sensation persisting after the stimulus has been removed. (c) An inability on the part of the patient to tell correctly the sequence of several pressure sensations rapidly repeated, a phenomenon due to a persistence of the foregoing impression which, in consequence, runs into the succeeding one. (d) The rapid disappearance of a painful sensation, though the stimulus is maintained. It has frequently been observed that the ability to recognise thermal stimuli is retained a long time in tabes, and in my experience cold impressions can be distinguished after a hot body has ceased to excite a corresponding sensation, although Topinard enunciated the reverse opinion.

Diminution of the muscular sense is seldom absent. This impairment may be general or limited in its distribution. When pronounced, the

patient is unable to recognise the position of his legs in bed; he has lost the power of distinguishing between weights; and the strongest faradic current may fail to excite pain on contracting the muscles (muscular analgesia).

The motor disturbances display themselves most frequently in the lower extremities, and hence are witnessed as deranged phenomena of station and locomotion. The impairment consists essentially in motor inco-ordination, and not, as was thought before the time of Todd, in paralysis. Thus, as Duchenne showed, the impaired style of walking is due to a want of certainty and precision in the performance of combined and complicated movements, the *muscular power being practically undiminished*.

The inco-ordination is noticeable in the way in which the patient stands (static ataxia), as well as in his style of walking. When placed in the so-called "ataxia position," *i.e.*, with the feet close together and the eyes shut, he experiences a difficulty in maintaining the erect posture, having, when the case is slight, to exert himself in order to preserve his balance, whilst in well-marked examples of the disease he reels and falls at once.

The gait varies very much in different cases. In some, very careful observation is necessary in order to detect any anomaly; in others, it is the prominent feature of the case, and at once suggests

a diagnosis. Before the stage of fully developed ataxia it may be observed that the patient cannot walk in a straight line, especially if he raise his eyes from the ground or close them. His attempts to do so are marked by staggering and unsteadiness which cause him to deviate from the prescribed line. He also experiences a difficulty in turning, which he does after the fashion of the pantaloons in a pantomime — *i.e.*, in a shuffling and uncertain manner. In some cases of undoubted tabes, however, the gait is in no way removed from the normal, except so far as it might resemble the gait of a person who has not walked for some time—*e.g.*, a patient convalescent from an acute disease. When the ataxic gait is very pronounced, the patient is quite unable to walk unless his eyes are directed with the utmost vigilance to the part of the floor where his feet are, and even then the movements of his legs are disorderly, and he can only proceed with the aid of a couple of sticks. As the passive leg is being brought forward it is raised up and thrown in advance in an exaggerated manner, and then the heel is brought down to the ground with unnecessary violence. The patient is always embarrassed when walking on ground to which he is unaccustomed, and the inco-ordination is increased tenfold when he closes his eyes or attempts to walk in the dark. Even in this stage fatigue may be foreign to him, and a walk of a

few miles may be executed with comparative comfort. The muscles are soft and flaccid, and entirely free from rigidity until very late, when true paralysis and contractures usually supervene.

The electrical reactions are not important; in the earlier stages there is seldom any departure from the normal, but in advanced cases a diminution of the electric contractility is the rule.

Although motor paralysis is by no means common in locomotor ataxia except as a feature of its closing scenes, it not unfrequently happens that the patient's legs will suddenly give way when walking or standing, and he may fall to the ground; in other words, a transient motor paralysis has overtaken him. In some cases this paralysis may last for days before it passes off. Dr. Buzzard has especially called attention to such attacks.

One of the earliest and most important features is the absence of the patellar-tendon jerk. The importance of this sign, first described by Westphal, cannot be estimated too highly, for though the knee-jerk is present in some few cases of tabes, and is physiologically absent in a few people, its absence, nevertheless, in a patient whose quadriceps femoris muscles are not paralysed and exhibit idio-muscular* contractility is very suggestive of the disease.

* *i. e.* Contraction of a muscle as the result of a sharp stroke, say with the finger.

There is nothing constant in the condition of the ordinary skin reflexes; very often the plantar is increased for some years, whilst it may be impaired early in the case; usually, however, there is an active reflex in one or other of the situations where they are wont to be manifested.

Micturition is seldom performed in an entirely normal way. At first there are generally symptoms of irritation of the bladder; later on, the patient may experience a difficulty in emptying the bladder, standing usually a considerable time before the process is commenced, and even then the urine runs away slowly. Subsequently there is dribbling from paralysis of the detrusor, and there may be almost complete vesical paralysis in very advanced cases.

There is often constipation; and the rectum is generally the seat of very uncomfortable sensations, such as shooting pains, a sense of fulness, with a constant desire to pass flatus, &c., but there is very seldom paralysis of the sphincter.

The eye furnishes a series of interesting symptoms. In the first place the pupil often undergoes a change, and this alteration may occur as an altered reflex (the Argyll-Robertson pupil), as myosis, or as a dilatation of the pupil. The first two of these conditions are very common. In the Argyll-Robertson pupil the stimulus of light fails to induce any contraction, whilst the pupillary

movements are active during efforts of accommodation—*i.e.*, accommodation for a near point is attended by contraction. Associated with this phenomenon will often be observed the loss of another intra-ocular reflex, *viz.*, the pupil will no longer dilate when the skin of the front of the neck is irritated, as it will in health.

Myosis, or contracted pupil, occurs in more than half the cases, and is observed in both eyes, though generally more marked in one than the other; this symptom is probably due to paralysis of the cilio-spinal nerves. Mydriasis, or dilated pupil, is much less frequently met with.

The ocular muscles are very often paralysed, resulting, in slight cases, in diplopia, and in more severe ones in marked deviation of the axes. The third and the sixth are the nerves that are most frequently affected. In many cases the squint or diplopia are but transient phenomena, and frequently mark the very commencement of the case.

Atrophy, or gray degeneration of the optic nerves, also occurs with tolerable frequency. This morbid condition produces blindness, and may develop in the early stages of the disease. The altered disc may easily be recognised by the ophthalmoscope.

Disturbances of hearing, taste, and smell may also be observed. The auditory nerves, according to Pierret, are often affected, whilst anomalies of

taste and smell are nearly always associated with disturbances of the trigeminus.

Not unfrequently there are marked disturbances of the sexual functions. These, when they take the form of irritation and prompt the patient to excesses, and when they occur early, as they often do, are frequently regarded as the exciting cause of the affection. In other cases sexual weakness manifests itself from the first, and in all is sure to develop as the case advances, though excessive desire and full sexual power have been observed to last for many years in not a few instances.

Trophic changes make their appearance in the bones and skin. The affections of the joints which Charcot described occur at a comparatively early stage; the ends of the bones which enter into the affected joints become atrophied, and deformities arise in consequence. The knee-joint is most frequently affected, and after it the shoulder, the elbow, and the hip. Bed-sores are late complications, but skin eruptions such as herpes and different forms of bullæ may appear from time to time, and, as Buzzard has shown, are sometimes related to the attacks of shooting pains.

PHYSIOLOGICAL INTERPRETATION OF THE SYMPTOMS.

The shooting pains are explained by irritation of the posterior nerve roots, and, according to Buzzard, the gastric crises depend upon irritation

of the nuclei of the vagus by sclerosis. That the staggering gait and muscular inco-ordination generally are not due to impairment of ordinary sensation is shown by the fact that, according to the clinical records, there is no connection whatever between the degree of anæsthesia and the motor impairment. On the contrary, it is probable that the inco-ordination results from interference with cerebello-afferent fibres in the posterior root-zones, or, in other words, from disease of those fibres whose function is to co-ordinate afferent impressions before they are forwarded to the higher centres.

The common sensory disturbances are the necessary outcome of sclerosal atrophy of the posterior roots and gray horns; and the later muscular atrophy, paralysis, and contractures owe their origin to an extension of the lesion to the anterior gray horns and lateral columns.

PROGNOSIS AND DURATION.

With but few exceptions locomotor ataxia is a disease which runs a long course. Sometimes, though rarely, the symptoms develop and succeed one another rapidly; the patient is early bed-ridden, and may succumb in a few years, but it is quite a common event for patients to last 25 and even 30 years. In a well-marked example at present in the Newcastle Infirmary, the patient is

completely bed-ridden, and the symptoms are only of eight months' duration. Most of the cases end in death, but a few have resulted in recovery.

TREATMENT.

Anti-syphilitic remedies appear to do very little good, and, indeed, harm may be done by an injudicious administration of mercury. The most useful remedies are the nitrate and phosphate of silver, phosphide of zinc, and cod-liver oil.

Hydro-therapeutics may be of service as carried out in a well-conducted and salubrious institution.

Galvanism is useful in relieving the lancinating pains, and should be applied to the limbs as well as to the spine. Bromide of potassium is often employed for the same purpose, and recently salicylate of soda has been recommended, but should either fail resort must be had to morphia. In some cases the sciatic nerves have been stretched with marked benefit, especially as a treatment of the pain. I have only adopted this method in two cases, in both with decided success,

CHAPTER XXXVII.

LATERAL SCLEROSIS

(Spastic Spinal Paralysis).

THREE forms of sclerosis of the lateral columns have been recognised—primary, amyotrophic, and secondary or deuteropathic. The first two are primary lesions; the last is a descending degeneration, and implies an antecedent focus of disease from which the sclerosis or degeneration spreads downwards as a consecutive lesion.

PRIMARY LATERAL SCLEROSIS.

MORBID ANATOMY.

(See figs. 36, 37, and 38, p. 255).

The pathology of this very rare affection is obscure, and up to the present time exceedingly few undoubted cases have been recorded. The characteristic lesion of the disease consists in symmetrical sclerosis of the pyramidal tracts of the lateral columns, a limitation of the morbid process which a careful examination of the cord very seldom reveals. So often, indeed, has the post-mortem examination failed to confirm the clinical diagnosis of a primary symmetrical lesion confined to the lateral columns, that some have been

tempted to deny its existence. The morbid process has its origin, it is supposed, in the interstitial substance of the pyramidal tracts, the nerve tubes suffering a secondary impairment.

ETIOLOGY.

The etiology is by no means understood. Some regard exposure to cold, injuries to the spine, and syphilis as exciting causes. Bétous has suggested that lead impregnation may induce the disease, but the whole subject still remains in a state of uncertainty. Nor are the predisposing causes any better known. The cases seem to occur most frequently in middle life, and men are more often affected than women. The instances of lateral sclerosis that have come under my observation in children have all appeared to me to be examples of secondary descending sclerosis.

SYMPTOMS.

The cases are usually slow in developing, and run a very chronic course. As the lesion is symmetrical and confined to the pyramidal tracts, and commences in the lower portion of the cord, the phenomena are observed in both legs, and consist almost entirely of motor and reflex disturbances, due respectively to an impairment of the motor tracts and a blocking of the inhibiting impulses that pass from the cerebrum to the reflex centres in the cord.

The onset is gradual, and manifests itself by a sense of weakness and fatigue in the lower extremities which interferes with walking. Stiffness is felt, and before very long the muscles are observed to be firm, and show a tendency to rigidity. The increased muscular tension is accompanied by increased knee-jerk and ankle-clonus, signs which are amongst the early phenomena of the disease. After a time the muscular rigidity becomes very prominent, and the patient is unable to flex his limbs when they are in the straight position, though his motor power may be only partially lost. The gait then becomes distinctly *spastic*. Owing to the permanent extension of the leg, the foot being somewhat extended, the knee of the passive leg cannot be flexed as it is being advanced, consequently the toes are scraped along the ground and are made to describe an arc of a circle, the advance being aided by rotation upwards of the pelvis towards the side of the leg on which the body is resting. Thus the walk is more or less waddling. As the stiffness increases twitchings and tremors often make their appearance, and the patient is totally unable to walk or stand. The tendon-jerk phenomena become greatly increased, and, as a rule, the plantar reflex is slightly, and in some cases markedly, increased, though it is often diminished.

Sensory disturbances are, until very late, en-

tirely absent. The bladder and rectum in general escape, but some patients experience a difficulty in starting the process of micturition, or they may be unable to empty the bladder completely. The nutrition of the muscles is not affected for a very long time; but during the later period of the case when the lesion has extended to the anterior cornua, the muscles atrophy, the paralysis increases, and the rigidity passes off, whilst the reflexes are diminished.

When the disease affects the upper extremities, which it generally does sooner or later, they become stiff and rigid, and in time contracted.

Some few cases have been known to recover, but patients suffering from this disease generally linger on for many years, remaining almost stationary, or advancing very slowly, until they are cut off by some intercurrent affection.

DIAGNOSIS.

The motor paralysis with the rigidity and increased knee-jerk, point at once to the lateral columns; but the great difficulty lies in drawing a distinction between primary and secondary sclerosis. In making a diagnosis, stress must be laid upon the fact that in the primary affection the motor weakness is accompanied in its march, almost from the commencement, by spasticity of the muscles, the rigidity becoming soon a more

prominent symptom than the paralysis. In secondary degenerations the rigidity is generally preceded by paralysis for some time. Again, in primary sclerosis there are neither bladder nor sensory disturbances; in the secondary form, on the other hand, the bladder is frequently affected and there are very often pains, girdle sensation, and anæsthesia. And lastly, the absence of trophic disturbances of the skin (bed-sores) and muscles is in favour of the primary lesion; but it must never be forgotten that it is one of the rarest of spinal affections.

TREATMENT.

Hydropathy (cold-water cure) may be tried; and the continuous current passed through the diseased portion of the cord. Erb speaks well of both measures.* As internal remedies, cod-liver oil and nitrate or phosphate of silver have been recommended, with iodide of potassium and mercury in syphilitic cases.

AMYOTROPHIC LATERAL SCLEROSIS.

A few remarks upon the morbid anatomy and symptoms of this interesting but rare affection will suffice. Charcot, who first described the

* Ziemssen : "Cyc. of Pract. of Medicine," vol. xiii., p. 645.

disease, had his attention drawn to certain cases that suggested the union of progressive muscular atrophy with lateral sclerosis. In other words, these cases were marked by atrophy and paralysis, the paralysed muscles becoming rigid as they diminished in bulk. The pathological features were proved to consist of primary symmetrical sclerosis of the pyramidal tracts of the lateral columns, along with degenerative atrophy of the anterior gray horns. The lesion is most pronounced in the cervical enlargement, though it invades the motor tracts and some of the ganglionic cells of the medulla also, and ultimately descends the cord to the lumbar enlargement.

Consecutive morbid changes develop in the anterior roots and peripheral nerves, the majority of the nerve tubes undergoing a certain degree of simple atrophy. The nuclei of the medulla most frequently affected are the facial, spinal accessory, and hypoglossal.

Cases of amyotrophic lateral sclerosis usually run a rapid course; indeed, the patients seldom survive three years, and may, as Charcot tells us, succumb within the year.

The recorded examples of the disease have occurred in middle life, between 25 and 50; and the majority have been women. A few have been ascribed to exposure to cold, but the etiology is not understood.

In the majority of cases the upper extremities are first affected. Numbness is complained of, and the muscles become weak, atrophied, and painful on pressure; but, in addition, it is important to recollect that rigidity makes its appearance *pari passu* with the paralysis and wasting. Before long the arms become markedly crippled; contractures occur, and the arms are pressed against the body, the forearms are partially flexed and pronated, and the fingers are closed within the hands which are flexed at the wrist. Subsequently the lower extremities become affected. Here motor paralysis first makes its appearance, the enfeebled muscles becoming rigid by degrees, but without any diminution in their bulk.

A comparison between the arms and legs at this stage might suggest two very different affections, but the observer is not left long in doubt as to the identity of the lesions, as the paralysed and rigid legs soon begin to waste, and then to contract. In time, symptoms of bulbar paralysis manifest themselves, the development of which phase of the disease the patient does not long survive.

It will be seen from the above brief description that the disturbances are almost entirely motor. There is an absence of anæsthesia and bed-sores, and the vesical and rectal functions continue to be performed in a normal way. Ten-

don-jerk phenomena are increased at first, but become lessened as the atrophy progresses.

No treatment appears to be capable of arresting the disease, the prognosis being exceedingly grave.

The subject of secondary lateral sclerosis, or secondary descending degeneration of the pyramidal tracts of the lateral columns has already been discussed (p. 254).

CHAPTER XXXVIII.

INHIBITORY PARAPLEGIA

(Hysterical Paraplegia).

RECOGNISING with many others who have been called upon to treat cases of aggravated hysteria, much that is unsatisfactory in the term "hysterical," I have latterly adopted the word *inhibitory* in its place—especially with respect to the paralyses, for the following reasons:—In the first place, I am strongly of the opinion that the word "hysteria" should be eradicated from professional terminology, and its employment by the public discouraged as much as possible, for I am persuaded that incalculable harm has been wrought through the flippant application of a word, absolutely meaningless in itself unless significant of weakness and intended as a reproach, to morbid states demanding most careful treatment. In the next place, having determined as far as possible to set aside the term hysteria, and seeking a synonym which would have the recommendation of being free from social abuse, whilst more or less suggestive of the pathology of the disorder, the word "inhibitory" occurred to me as being most suitable, and especially applicable to the more

common motor phenomena of hysteria. Without attempting to enter at all fully into the nature of this functional derangement to which the nervous system, especially of women, is so much exposed, I may briefly explain what I mean by applying the term inhibitory to functional paralysis. It is an established fact in physiology that the basal masses of cerebral gray matter, especially the corpora striata and optic thalami, exercise an inhibitory influence over the cord centres, and that, in turn, the highest (cortical) centres exert a similar repression over the basal ganglia. Now, suppose the incitations of unconscious cerebration, in this case the inhibitory influences of the cortex, are no longer active, the uncurbed basal ganglia will exercise such a degree of inhibition upon the subordinate centres in the cord as to paralyse them, and thus produce the motor derangement.

Inhibitory paralysis occurs in a great variety of forms of which the paraplegic is the commonest, the most difficult to diagnose, as well as the most prone to last, and therefore it is the most worthy of notice in a book that purports to treat of organic disease of the brain and cord.

By far the greatest proportion of the cases of inhibitory, or hysterical paraplegia occurs in women between the ages of sixteen and forty-five; a few occur in boys about the period of

puberty, but in their case the disease is not manifested in a typical form, and, as a rule, soon passes off.

In some cases the onset is sudden, and the paraplegic state is fully established at once; but in others, again, the paralysis is gradually developed, and backache with "tender spine," anomalous pains in the legs, and weakness in one or both lower extremities are complained of for some time before any pronounced paralysis shows itself. In my experience severe and obstinate cases often commence gradually. In one case—a most intractable one—the prodromal symptoms lasted two and a half years before the paraplegia became pronounced; in another, also a most aggravated case, the patient became completely paralysed in the lower extremities two months after the symptoms first presented themselves. The gradual onset is much more common than the sudden.

A typical, exaggerated case when seen as soon as the paralysis is fully developed presents the following features:—The legs are almost completely paralysed, both as regards motion and sensation,—tactile sensibility may not be completely abolished, but painful impressions rarely, if ever, persist. The limbs are rigid and extended; there is absolutely no wasting, and the muscles show a normal electrical excitability. There are no bed-sores; the urine is never alkaline, and is generally passed normally

though there may be retention, but there is never paralysis of the vesical sphincter. The plantar reflex is completely abolished, whilst there is marked ankle-clonus and knee-jerk. There is nearly always analgesia to be discovered in other situations than the lower extremities; in many cases whilst the skin of the paralysed extremities is completely analgesic the muscles are not so, as pain is often complained of when they are tetanised by a strong faradic current, and it is not unusual to discover that the patient can recognise thermal, though not tactile or painful stimuli; there is nearly always pain in the back with areas of hyperæsthesia scattered along the vertebral column. And, finally, the patient appears to subsist upon a marvellously small quantity of food.

The subsequent history of the case is very varied. In some few instances there is a rapid recovery, even without any special treatment; others, again, run a protracted course, and the patients may be invalided for years, if not for life. A short while ago I had the opportunity of visiting one of the best Hospitals for Incurables in the kingdom, where there were a large number of interesting spinal cases including about half a dozen examples of complete functional paraplegia, and in several instances the patients had been upwards of ten years in the hospital. As the case pro-

gresses the legs waste, the rigidity passes off, the knee-jerk and ankle-clonus become less pronounced, and generally the analgesia and other sensory disturbances abate in intensity. It must be borne in mind, however, that it may take years to bring about these changes.

In many cases the motor paralysis is far from complete, and the patient may be able to move her legs in bed, or she may be able to walk dragging her feet, and aiding herself by crutches or by grasping the adjacent pieces of furniture. But even here there is marked analgesia, more or less tendency to rigidity, and increase of knee-jerk.

DIAGNOSIS AND PROGNOSIS.

Inhibitory paraplegia must be distinguished from primary lateral sclerosis, slow compression of the cord, and myelitis of the lumbar enlargement. Cases in which the legs become at once rigid and paralysed cannot be confounded with any of these affections, but it is often difficult to determine the nature of a case in which the onset has been slow. Primary lateral sclerosis is exceedingly rare, and is never attended by the exaggerated analgesia that is so characteristic of the functional attack. In compression myelitis—generally the result of Pott's disease—there are characteristic pains (*see p. 295*), and other signs of compression

of the cord; the sensibility is seldom early affected, and there are often bladder disturbances, and increased plantar reflex. Extensive myelitis of the lumbar enlargement such as would be likely to be confounded with a pronounced functional paraplegia is seldom difficult to recognise, for bladder disturbances, bed-sores, and wasting of some of the muscles,—generally features of the former affection, are absent in the latter.

The prognosis is undoubtedly good; in fact, cases that are properly treated always recover. In one case under my care the patient has been bed-ridden for more than four years, but she has at length commenced to improve.

TREATMENT.

In the first place it is essential to remove the patient from unfavourable social influences, the operation of which appears to promote the morbid condition which underlies the derangement. Thus, for example, it may be necessary to send her from home under the care of a nurse. Muscle-rubbing, which is a combination of muscle-kneading and shampooing, will be found to be of the greatest possible benefit. The muscles should be thumbed, rolled, and rubbed, beginning from the toes and ascending to the trunk. The spinal column ought also to be shampooed, and it is well before commencing the process to pour hot water along the

spine, the patient lying on her face. A daily use of medical rubbing should be insisted upon. Faradism should accompany the *massage*, and tonics may be administered, such as iron and strychnia, but bromide of potassium seldom does any good.

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